NATIONAL INSTITUTE OF SIDDHA

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THE TAMIL NADU DR. M.G.R. MEDICAL UNIVERSITY, CHENNAI – 32

PRE CLINICAL AND CLINICAL STUDY ON

AZHAL KALLADAIPPU

And The Drug of choice is

KARPOORA SILASATHU PARPAM

(DISSERTATION SUBJECT)

For the partial fulfillment of the

Requirements to the Degree of

DOCTOR OF MEDICINE (SIDDHA)

BRANCH I - MARUTHUVAM

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INTRODUCTION

The word "**siddha**" comes from the word '**siddhi**' which means 'An object to be attained' or 'perfection' or 'Heavenly Bliss'.

The siddha system of medicine, which is one among the oldest and foremost indigenous medical system. Siddha is a significant part of Tamil's culture and tradition due to its deep roots of Dravidian origin. More than just a medical system, siddha is a system dealing with intense spirituality and immense possibilities for the betterment of human being. Unlike other systems, siddha system aims in both the treatment and prevention of the disease.

"அண்டத்தி லுள்ளதே பிண்டம்
பிண்டத்திலுள்ளதே அண்டம்
அண்டமும் பிண்டமு மொன்றே
அறிந்து தான் பார்க்கும்போது"

-சட்டமுனி ஞானம்

Man is said to be the Microcosm, and the world the Macrocosm, because what exists in the world exists in man. So, man must be looked upon as an integral part of universal nature. Further, corresponding forces acting in and through the organisms of the world. This closely related to the 96 constituent principles.

Nature is the material cause not merely of the outer Universe but also of our body with all its grosser and subtler divisions and components. The human body is composed of ninety-six principles in nature including elements, bodily and mental organs, faculties, matter etc,

The world in which the above three processes take place is made up of five basic elements viz., Earth, Water, Heat, Air and Ether. And the man is capable of identifying all the objects of this world only through his five sense organs involving five basic elements.

According to the Siddha system, various psychological and physiological functions of the body are attributed to the three humours viz., Vatham, Pitham and Kabam represent respectively the air, the fire and the water and seven thathus, first is Saram responsible for growth, development and nourishment, second is Cheneer responsible for nourishing muscles, imparting colour and improving intellect, the third is Oon responsible for shape of the body, fourth is Kollzuppu responsible for lubrications, fifth is Elumbu responsible for body structure and posture and movement, sixth is Moolai (Brain) responsible for strength, and the last is Sukilam responsible for reproduction, which form the connection link between Microcosm and Macrocosm.The three humours when deranged, they bring about diseases peculiar to their influence.

Siddhar classified diseases occurred to human beings into 4,448, they also had many interventions for each disease due to the derangement of three vital humours.

According to **Tamil vaithyasathagam**, the pingalai, urinary bladder, stomach, umbilical, epigastric region, sweat, saliva, essence of food, eyes and skin are the places where pitham sustains.

தானான பித்தம் பின்கலையைப்பற்றிச்
சாய்வான பிராணவாயு வதனைச் சேர்ந்து
ஊனான நீர்ப்பையி லணுகி மூலத்
துதித்தெழுந்த வக்கினியை யுறவு செய்து
மானேகே ளிருதயத்தி லிருப்பு மாகி
கோனான சிரந்தனிலே யிறக்க மாகிக்
கொண்டு நின்ற பித்தநிலை கூறினோமே.

- தமிழ் வைத்திய சதகம்

The disease called kalladaippu is caused due to derangement of pitha humour.

In siddha text, Siruneer noi or Moothira noi, this is classified into,

1. Neerinai arukkal noi
2. Neerinai perukkal noi

This has been mentioned by Therayar in his **'Therankarisal'** as follows,

"நீரிரு வினைக் குணத்தை நீயறிவித்துச் சொல்வாம்
நீரினைப் பெருக்கலொன்று நீரினை யருக்கலொன்று
நீரிழிவுடனே கொல்லும் நீர்கட்டு வினைகளொன்று"

-தேரன் கரிசல்

The disease **kalladaippu** is placed under the Neerinai arukkal noi.

As per Agathiyar Rathina Churukka naadi kalladaippu is classified into 80.

"அஞ்சாகுங் கல்லடைப்பு எண்ப தாகும்"

-அகத்தியர் இரத்தினச் சுருக்க நாடி

Kalladaippu is described well in the classical siddha text **Yougi vaithiya chinthamani**. It is classified into four types and **azhalkalladaippu** is one among them, which is described by Yougi muni.

"தோன்றினதோர் நாலினிட நாமங் கேளாய்
பூன்றியதோர் பித்தத்தின் கல்ல டைப்பு

-யூகி வைத்திய சிந்தாமணி.

The clinical features of Azhal kalladaippu may be correlated with that of Renal Calculi in modern science. It includes the symptoms like oliguria, urethral pain mimics a pain caused by an insertion of hot iron in the urethra, sweating all over body, anuria, agonizing pain, blood stained calculus stagnated in urethra.

Kidney Stone disorders are common in men than in women. Majority of the patients are between the 20-55 years of age. The highest incidence of kidney stone is in 30-45 years of age group and the incidence declines after the age of 50 years of age. It affects 10-12% of the population in industrialized countries.

Epidemiology of Renal calculi varies according to the geographical areas and socioeconomic conditions. Renal calculi occurs in all parts of the world, with a lower life time risk of 25 percentages in Asia 3-15 percentages in the west 20 percentage in India.

Global climate change is the environmental factor that affects stone disease rates according to research presented at the 103rd Annual Scientific Meeting of the American Urological Association (AUA) and based on the effects of global warming, the percentage of people living in areas designated as high risk for kidney stone formation would increase from 40% in 2000 to 56% by 2050, and up to 70% by 2095. This would result in a significant "climate-related" increase in kidney stone events.

Renal calculi can be prevented by the most important thing into drink plenty of water daily the goal should be to urinate from two to four liters per day make sure you avoided getting dehydrated, there are no specific dietary recommendation until a stone from your system has been analysed. After analysis diet can be evaluated and changes recommended.

Although the surgical techniques have taken greater strides, yet the common man in developing country like India may not find it affordable. Hence the formulation of **KARPOORA SILASATHU PARPAM** described in siddha text, Agathiyar chendhooram 300, as that of siddha medicine for the management of **kalladaippu**. The mode of preparation seems to be simple. The main Ingredients formulation is found to possess Lithotriptic and Diuretic effects. The above said drug formulation has not undergone any clinical trial so far. Hence I have selected the siddha formulation **"KARPOORA SILASATHU PARPAM"** for further clinical evaluation in **AZHAL KALLADAIPPU** noi.

AIM AND OBJECTIVES

AIM:

To document the siddha drug **Karpoora Silasathu Parpam** in the treatment of **Azhal Kalladaippu** (Renal Calculi) by the standard process of evaluation of safety and efficacy of the drug.

OBJECTIVES

PRIMARY OBJECTIVE:

To evaluate the therapeutic efficacy of siddha drug **Karpoora Silasathu parpam (Internal)** in the treatment of **Azhal Kalladaippu (Renal Calculi).**

SECONDARY OBJECTIVE:

1. To evaluate the safety profile (acute, long term toxicity studies) of this drug.

2. To study the effect of other co-factors such as age, sex and siddha parameters.

அழல் கல்லடைப்பு

Kalladaippu is described well in the classical siddha text Yougi vaithiya chinthamani. It is classified into four types and **azhal kalladaippu** is one among them, which is described by Yougi muni based on three vital humours in our body.

"தோன்றினதோர் நாலினிட நாமங் கேளாய்
பூன்றியதோர் பித்தத்தின் கல்ல டைப்பு

-யூகி வைத்திய சிந்தாமணி.

கல்லடைப்பு - இயல் (DEFINITION)

According to the text of **Siddha maruthuvam (pothu) by Dr. Kuppusamy,**

There is gradual or suddenly obstruction to the flow of urine, pain with burning sensation in the urethral tract, Low back pain, renal angle pain and sand like crystal deposit in urine. These are characteristic features of **Kalladaippu**.

According to the text of **Jeevarachamirtham,**

Kalladaippu is defined as pain in and around the umbilicus, fever, dysuria and urine smelling like that of goat's urine.

According to the **T. V. Sambasivam pillai,**

Large concretions of minerals in the bladder or kidney produce calculus or gravel. It is attended with difficulty in passing urine.

According to the text of **Agathiyar gunavagadam,**

"தானென்ற மூத்திரத்தால் நறநறவென்று
தங்கியதோர் பொடியெனும் மணல்தானப்பா
வானென்ற சிறியதொரு கல்லா வதப்பா
வளமாக வந்துவிழும் நோய்க்குத் தானே
ஏனென்ற அச்மரிரோக மென்ற பேராம்
எளிதாக கல்லுக்குள்தான் விழுகும் போது

கோனென்ற குண்டிக்காய் மூத்திரக்குழலப்பா
குணமான மூத்திரப்பை நீர்தாரை கேளே
கேளடா முன்குறியில் எரிச்சல் கண்டு
கெடியாக வேதனைகள் காட்டுமப்பா
வாளடா சிறியதொரு கற்கள் தானே
வளமான மூத்திரப்பை குழல்வழிப் படியாய்த்
தேளடா வரும்போது திரே கந்தன்னில்
தெரிப்பது போல் யிருவேதனை செய்யூம்பாரு
நாளடா கற்கள் தானிறங்கி விட்டால்
நலமான வேதனைகள் தான் தீரும்பாரே"

-அகத்தியர் குணவாகடம்

Agathiyar says the definitions of Kalladaippu as sand like crystal deposited in urine, followed by small size of stones are excreted in urine. Stones are stagnated in kidney, ureter, urinary bladder and urethra. Pain with burning sensation start in urethral orifice followed to agonizing pain occurs during the stone moving in urethral tract from the bladder, when the stone removed pain also relieved.

நோய் வரும் வழி (ETIOLOGY)

“தெளிந்ததோர் கல்லடைப்பு உற்பத்தி கேளாய்
சிறிதுநாட் டொடங்கியே மூகந் தன்னால்
தளிந்ததோர் சலப்பையி லுதிரந் தோய்ந்து
சந்தசத் தாகவே பருத்துக் கொள்ளும்
வளிந்ததோர் வாதபித்தங் கோபித் தக்கால்
வந்துபெருங் கல்லாய்நீர் வழிய டைத்து
நளிந்ததோர் நாலுவிதக் கல்ல டைப்பு
நண்பான வரலாறு நாட்டக் கேளே”

- யூகி வைத்தியசிந்தாமணி

It is worthwhile to mention the poem of Yougi mamunivar who is authority of Siddhars regional and humoral pathology. He has revealed about this disease since 14th century.

Yougi mamunivar says that as blood clotted in urinary bladder due to urinary tract diseases followed by swelling of urinary bladder, urinary stones are formed in urinary tract induced by humour of vatham and Pitham.

"நாட்டமாய்க் கற்பழித்துக் கடமை வாங்கி
நலிபண்ணிக் கூடாமல் வழக்குப் பேசி
கூட்டமாய்க் குருவுடைய உடமை தன்னைக்
கொடாமலே கைக்கொண்ட கொடுமை யோர்க்கும்
வாட்டமாய் வம்புதம்புத் திரிந்த பேர்க்கும்
மாறுபாடா யெடுத்துப் பொருள்க டனைக்
காட்டியே கைக்கொண்டு கபடு பண்ணும்
காலாந்தர் கல்லடைப்பிற் காளாவாரே"

-யூகி வைத்திய சிந்தாமணி

In this explains that mind plays a major role in causing many diseases and connection between body and mind and soul is established i.e., the mukkutram deranged by internal factors such as sexual perversion, anger and robbery.

கலங்கினதோர் தண்ணீர்தான் குடித்த பேர்க்குங்
கல்லெலும்பு மயிர்மண்தான் கலந்தன் னத்தில்
அலங்கினதோ ரன்னங்க ளருந்த லாலும்
அழுகலோடு மூத்தபண்ட மருந்த லாலும்
மலங்கினதோர் மாப்பண்ட மருந்த லாலும்
மந்தத்தில் வாய்வான பதார்த்தந் தன்னை
துலங்கினதோர் ருசிதன்னிற் சுவைத்த லாலும்
சுருக்காய்க்கல் லடைப்புவந்து தோன்றுந் தானே

- யூகி வைத்தியசிந்தாமணி.

The causes mentioned here,

- Intake of turbid water
- Food contaminated with stones, bones, hair and sand
- Intake of deteriorated food stuff and starch substances
- Eating flatulence producing food while indigestion.

நீரினைத் தடுத்தல் செய்யின்
நீர்கட்டுத் துவாரம் புண்ணாம்
பாறிடுஞ் சந்து சந்தில்
பண்புறு நோவ தாகும்
நேரிலங் கயருஞ் காமியம்
நிச்சய நோதல் செய்யும்
பாரினி லபான வாயு
பண்புறச் சேரு மன்றே

-சித்த மருத்துவாங்கச் சுருக்கம்

Siddha maruthuvanga churukkam explained that urination is one of the 14 natural urges. When one suppress this visceral reflex it don't pass urine regularly, it will cause obstruction in the urethral passage, ulceration in the urinary tract, pain in the joints and genitalia and distension of the lower abdomen, urinary tract infection with ulceration in the genitalia and deranged of keezh nokkungaal. This leads to the formation of calculus.

The author also explains that ejaculation of semen is one of the 14 natural urges when one suppress this reflex it leads to fever, retention of urine which favours urinary calculi, chest pain, arthralgia, urinary infection, spermatorrhoea and white discharge.

சுக்கிலந் தனை யடக்கின்
சுரமுடனீர்க் கட்டாகும்
பக்கமாங் கைகால் சந்து
பாரநோய் வழியிறங்கும்
மிக்கமார் நோயுண்டாகும்
மிகுத்திடும் பிரமேகந் தான்
தக்கதோர் போதுமாகின்
தரித்திடும் வாயுக் கூறே

- வியாச பகவான் சரீர சூத்திரம்

According to T.V. Sambasivam pillai

A urinary disease occasionally developed in the urinary bladder, which is called vesical calculus. It is said to be due to the deranged Vayu encircling or prevailing in the region of the abdomen arising from any of the following causes viz:-

- Suppression of seminal discharge during sexual intercourse.
- Retention of semen in the spermatic region in involuntary discharge during nocturnal emissions due to excessive heat in the body.
- Prevention of discharge of semen induced by taking aphrodisiac preparations.

According to Noi vilakkam

"கரு நீரடக்கல் விரையில் அடிபடல்
நீரியந்தாக் கல் சிறுநீரடக்கல்
வளிநோய் மிருக்கு முணவும் ஒழுக்கமும்
கடைப் பிடித்திடுதல் மேகமுதற் பல
பிணியுறல் எழுமிவை யடிப்படையாகக்
கல்லடைப் பென்னுங் கடும்பிணி விளையும்
வளியது மீறியை யொடு மல்லாது
கருநீ ரொடுங் கலந்துற நீரகத்துச்
சிறுநீர்க் கழிவு தொகுத்தலாலும்
அன்னவை கல்லெனத் திரளு மென்ப"

- **நோய் விளக்கம்**

- Derangement of humour in blood
- Excessive indulgence in sexual activity or sexual perversion
- Trauma on testis
- Suppression of urine and semen
- Inflammation of bladder
- Syphilis (Mega noi)
- Stagnation of urine in urinary tract
- Dryness of semen causes the formation of stones
- Increased intake of food that cause flatulence

விழுகு சிலசேரம் விடுபட்டு நீரோடும்
ஒழுகிய வாயுவு மொதுகினால் நோகாது
வழுகிய மந்தத்தால் வாயுவந்தே புகில்
கழுகி முதிர்ந்திடும் கல்லடைப்பாகுமே

-திருமூலர் கருக்கடை வைத்தியம்

Stone is formed by derangement of humours of vatham and Pitham.

According to Saraga samhithai

சிறுநீர்ப்பையில் கல் தோன்றுதல்:

வாதம் சீற்றமடைந்து சிறுநீர்க்குழாயில் சேர்ந்து அதன் வழியை அடைத்து சிறுநீரை மட்டுமோ விந்து கலந்த சிறுநீரையோ நீர்த்தோற்றத்திலிருக்கும் பித்தத்துடன் கூடிய சிறுநீரையோ கபத்துடன் கூடிய சிறுநீரையோ உலர்ந்து போகச் செய்யும். அவ்வாறு வறண்டுபோன சிறுநீரில் கரைந்துள்ள பித்தம், கபம் அல்லது விந்து இவைகள் உறைந்து போவதால் பசுவின் பித்தத்தில் கோரோசனம் தோன்றுவது போல் கடினமான பொருள் தோன்றும். இது (அச்மரீ) கல் என்று கூறப்படும்.

-சரக சம்ஹிதை 3ம் பாகம்

சிறுநீர்ப்பையில் தோன்றும் கல்லின் இலக்கணம்

சிறுநீர்ப்பையில் தோன்றும் கல் கடம்பமலரைப் போல் தோற்றமும் நிறமும் கொண்டு கல்லைப்போல் கடினமாயும் வழவழப்புடன் முக்கோண வடிவில் மூன்று பொறைகளுடனும் மென்மையுடனும் இருக்கும். அது சிறுநீர் வழியையடைந்து சிறுநீர் வெளிவராமல் தடுத்து சிறுநீர்ப்பையில் கொடிய வலியையும் குத்தலையும் தோற்றுவிக்கும்.

POTHU KURIKUNANGAL

According to the text of Siddha Maruthuvam (pothu)

- Gradual or sudden obstruction to flow of urine
- Unbearable pain (agonizing pain) in the penis
- Excruciating pain and swelling is experienced at tip of penis if the calculus attempts to expel.
- Colicky pain radiating from loin to groin, lower abdomen, urethra and genitalia if the calculus is irregular with sharp projection.
- Burning and scanty micturition and haematuria.

According to the text of Aruvai Maruththuvam

- In starting stage, nausea with vomiting occurs
- pain in and around the umbilicus and penis
- Urine smelling like that of goat's urine
- Sometimes, blood appears while passing urine
- Sometimes urine passed from two ways.

SYMPTOMS ASSOCIATED WITH KALLADAIPPU

உக்கார சூலை

"குத்துமுக் கார சூலையின் குணந்தான்
கோர்வையாய் விலாவதனில் முதுகில் நெஞ்சில்
அத்தியில் நாபியினி லபான குதத்தில்
அதிகத்துன் மாங்கிஷந்தான் வளர்ந்து மேவிப்
பத்துமணற் பருக்கைபோற் சலத்து வாரப்
பதிநெருக்கி மூத்திரமாங் கிரிச்சி யுண்டாய்த்
தத்துசடங் கடுப்பெடுத்து மதிக லங்கித்
தளர்ச்சியொடு மயக்கமாய்த் தள்ளுந் தானே"

- யூகி வைத்தியசிந்தாமணி.

Excessive growth of muscles in chest region, back of trunk, umbilicus, anal and urethral orifice followed by stricture of urethral orifice, sand like crystals blocked in urethra. Dysuria, body pain, Impairment of conscious, tiredness and giddiness occur.

CLASSIFICATION OF KALLADAIPPU

தோன்றினதோர் நாலினிட நாமங் கேளாய்
சுருக்கான வாதத்தின் கல்ல டைப்பு
பூன்றியதோர் பித்தத்தின் கல்ல டைப்பு
புரண்டதோர் சிலேட்டுமத்தின் கல்ல டைப்பு
தீன்றியதோர் தொந்தமாங் கல்ல டைப்பு
தேகத்தைப் பற்றியே சிறிது காலம்
தான்றியே சலப்பையில் வந்தி ழிந்து
சருவியே லிங்கத்திற் றரிக்குந் தானே.

-யூகி வைத்திய சிந்தாமணி.

The most experienced of Siddhars, Yougi mamunivar who has studied the disease according to Regional and Humoral pathology classifies **Kalladaippu** into 4 types, there are

1. வளிக் கல்லடைப்பு
2. அழல் கல்லடைப்பு
3. ஐயக் கல்லடைப்பு
4. முக்குற்றக் கல்லடைப்பு

வளிக் கல்லடைப்பு (VALI KALLADAIPPU)

"தரித்துநா பிக்குங்கீழ்ச் சுருக்காய்க் குத்திச்
சலமலந்தான் வீழாமற் றம்ப மாகி
வரித்துமே லிங்கத்தில் வலியு மாகி
மருவியதோர் பொத்தியெல்லாஞ் சுரந்து கட்டி
திரித்தியே கிடக்கொடாப் புரட்ட லாகித்
தேம்பியே மூச்சுமாய் வயிறு முப்பும்
உரித்ததோர் சதைபோல உவர்ப்பு மாகும்
ஓங்கியதோர் வாதக்கல் லடைப்பு தானே"

- யூகி வைத்தியசிந்தாமணி.

Pain is felt just below the umbilical region and penis.

It is characterized by,

- Severe colic pain
- Dyspnoea
- Abdominal distension
- Oliguria
- constipation

அழல் கல்லடைப்பு (AZHAL KALLADAIPPU)

"அடைப்பாகிச் சலந்தானு மருவ லாகி
அயங்காய்ச்சிச் சொருகினாற் போலே
புடைப்பாகிப் பொத்தியெங்கும் புழுக்கமாகிப்
பூட்டுபோல் விகுவாகிப் புரட்ட லாகும்
மடைப்பாகி உதிரநிற மாய்க்கல் லாகி
வந்திழிந்து லிங்கத்தில் மாட்டிக் கொள்ளும்
குடைப்பாகிக் குற்றலாய்க் கூச்ச லாகிக்
குதட்டுமே பித்தக்கல் லடைப்புத் தானே"

- யூகி வைத்தியசிந்தாமணி.

In azhal kalladaippu, reduced urine output with characteristic burning sensation (similar to introducing a red-hot iron needle into the urethra), red blood coloured stones which blocks the ureter causing pricking pain and tenderness.

ஐயக் கல்லடைப்பு (IYYA KALLADAIPPU)

"தானான தொப்புளிலே வில்லுப் போலச்
சலியாமற் சுரந்துமே சற்றே குத்தும்
ஏனான காலொடு கைகள் சந்து
இடுப்புதான் குடைச்சலா யிசிவு காணும்
வேனான லிங்கத்தின் வேன்மை தன்னில்
விறுவிறென்றெ கடுப்பாகி வியர்வை யாகும்
தேனான வெளுப்புக்கல் சிறுகல் லாகச்
சிக்கலாய் வந்திறங்குஞ் சிலேட்டுமந் தானே"

- யூகி வைத்தியசிந்தாமணி.

Iyya kalladaippu is characterized by excruciating pain in the umbilical region, pain in the joints of upper and lower extremities, low-backache, spasmodic pain, sweating and gradual passing out of white coloured stone granules in the urine.

முக்குற்றக் கல்லடைப்பு (MUKKUTRA KALLADAIPPU)

"வந்திறங்கும் நீர்த்தாரை அடியிற் றானும்
மாவருத்த முண்டாகி வலியு மாகி
நொந்திறங்கி நீர்தானு மருவிப் பாயும்
நொய்தான சிறுமணற்போல் நொறுங்கிக் கல்லாஞ்
சந்திறங்கி நீர்வழியில் வந்து வீழும்
தாக்கான சிறங்கைக்கல் தினமொன்றுக்கு
நுந்திறங்கித் தினந்தினமு மிழிந்து கொல்லும்
தொந்தமாங் கல்லடைப்புச் சூட்டிட் டாயே"

- யூகி வைத்தியசிந்தாமணி.

In Mukkuttrak kalladaippu, severe pain is felt just below the urethral region with excess urination. It is characterized by disintegration of stones into small, sand like granules in the urine.

CLASSIFICATION ACCORDING TO NOI VILAKKAM

"வளி முதல் மூன்றினுந் தோன்றலாலும்
கருநீர் தன்னிற் தோன்றலாலும்
கல்லடை நால் வகைப் படுமெனமொழியே"

- நோய் விளக்கம்

There are four types of **Kalladaippu** according to Noi vilakkam

1. Vali kalladaippu
2. Anala kalladaippu
3. Iyya kalladaippu
4. Karuneer kalladaippu

வளிக்கல்லடைப்பு (VALI KALLADAIPPU)

"படர்மிகப் படுத்தல் பற்கள் கடித்தல்
நடுங்கல் உந்தியும் குறியும் பிசைதல்
கசடுகீழ் சளியொடு கழலல் அழுதல்
சிறுநீர் துளித்தல் என்பவும் பிறவும்

வளியின் கல்லடைக் குறியென மொழிய
கறுத்துஞ் சிவந்தும் முனைகள் பரந்தும்
வளியின் கல்லது வடிவுனு மென்ப"

- நோய் விளக்கம்

- Tongue biting, palpitation and shivering
- Crushing of the lower abdomen and genital organs
- Dribbling of urine
- The stones are blackish red colour

அனலக்கல்லடைப்பு(ANALA KALLADAIPPU)

"சுட்டென நீரியம் மிகவெம்பிடுதலும்
நோதலும் அவைக் கல்லடைக்குறியே
சிவந்துங் கறுத்து மஞ்சளாகியும்
சேங்குரு வடிவில் கல்லது தோன்றும்"

- நோய் விளக்கம்

- Burning micturition
- Dysuria
- The stones are reddish black or yellow in colour and passing of small stones

ஐயக்கல்லடைப்பு (IYYA KALLADAIPPU)

"நீரியங் குத்தல் திணித்தல் குளிர்த்தல்
எனுமிவை ஐயக் கல்லடைக் குறியே
வெளுத்தும் தேனிறமாகிய மொளிர்ந்தும்
பெரு வடிவுடைத்தாம் ஐய கல்லடைப்பு"

- நோய் விளக்கம்

- Pricking pain, forceful pain with severe intensity when passing urine
- Fever with rigors
- White or honey coloured shining or luminant large size stone expelled.

கருநீர் கல்லடைப்பு (KARUNEER KALLADAIPPU)

"கரு நீர்க்கல்லின் வளி சினந்தெழுந்து
விரைகளி னடுவில் அதுதனைத் தடுத்தலின்
கருநீர்க் கல்லடை மருவிடு மென்ப
நீரியம் நோதல் சிறுநீர் தடைபடல்
விரை வீங்கியிருத்தல் எனுமிவை பிறவும்
கருநீர் கல்லடைக் குறியென மொழிய
கருநீர்க் கல்லினை வளியது முடுகிச்
சிறியவும் பெரியவுந் துண்டுகளாக நொறுக்கிடும்
அவை சிறுநீர் வழி வெளிப்படவாகும்
அவை சிறுநீரினைத் தடுத்தல் நிற்கும்
சாற்றிய நீரினைத் தடுத்து நிற்பின்
ஆற்றல் குறைதல் வயிறு நோதல்
சுவைகெடல் வெளிறு மறுப்பு நீர்வேட்கை
வெல்வளி யெனுமிவை விளைந்திடு மெந்ப
சிறுவர்க் காயின் கல் சிறிதாதலின்
கருவியி னெடுத்தல் எளிதா மென்ப
கருநீர்க் கல்லடை சிறுவர்க் கில்லை
விரையும் உந்தியும் மிகைப்பட வீங்கல்
சிறுநீர் தடைப்படல் நோவு மிகுதல்
என்பவை துணிகல் மணலிடைத் தோன்றின்
பிழைத்த லரிதெனப் பேசுவர் புலவர்"

- நோய் விளக்கம்

- Sudden or gradual obstruction to flow of urine
- Excessive vali kutram breaks the stones into small and large size crystals and expels along with urine
- Sudden stoppage of urine stream
- Retention of urine
- Abdominal pain
- Loss of taste, excessive thirst
- Pricking pain with swelling of abdomen and testis
- Retention of urine or anuria may leads to renal failure and fatal

CLASSIFICATION IN DHANVANTHIRI VAITHYAM

"திருந்திய வாதபித்தச் சிலேற்பனம் பிரகோபத்தால்
வருந்த சுமரித்தா னான்கு வகைபடுங்கல்லெரிப்பான்
பிரிந்திடுஞ் சிலேற்பனா சுமரி பித்தாபின்னு
மிருந்திடு சுக்கிலாசுமரி நான்கு மெய்துமென்றே"

-தன்வந்திரி வைத்தியகாவியம்

In Dhanvanthiri vaithyam, **Kalladaippu** is classified into four types, they are

1. கல்லெரிப்பான்
2. சிலேத்தும அச்மரி
3. பித்த அச்மரி
4. சுக்கில அச்மரி

கல்லெரிப்பான் (KALLERIPPAN)

"கத்துநீர் நாளந்தன்னில் சுக்கிலந்தனிற் சிலேற்பம்
பித்தமீ துவர்த்தல் கல்லாய்ப் பிசகினீரடைத்து கொள்ளுங்
கொத்துநீ ரிற்றுவிழுங் கொப்புள்நோ குடம்புகாயுஞ்
சித்தமா யருசியுண்டாஞ் சேர்ந்த கல்லெரிப்பாமே"

-தன்வந்திரி வைத்தியகாவியம்

- Increased Iyyam and Azhal kutram dries the urine and semen forming calculi
- Sudden or gradual obstruction in urinary tract
- Dysuria
- Pain in umbilicus
- Fever
- Anorexia

சிலேத்தும அச்மரி (SILETHUMA ACHMARI)

"நீர்வரு நாளந்தன்னில் நின்றநீர் சிருத்துக்கொண்டு
சோர்தரும் சிலப்பு வெண்மை சுக்கிலம்போல்வீழும்
பேர்பெற நாலா மெட்டுப் பின்னமாய்க் கல்லுவீழும்
ஏர்பெறு சிலேற்பனத்தின் அசுமரி என்னலாமே"

-தன்வந்திரி வைத்தியகாவியம்

- Calculus in the ureter or urethra causes hydronephrosis
- Oliguria
- Reddish white in colour and falls out like semen
- Stones are expelled as 4 or 8 fragments

பித்த அச்மரி (PITHA ACHMARI)

"செய்யும்நீர் நாளந்தன்னில் பித்தத்தா லெரிப்பெல்ந்து
செய்யுவுஷ்ணத்தால் வெந்து சேங்கொட்டைபோல் கல்லுண்டாம்
நய்யவே தனைகள் செய்யும் நவில்குணம் பித்தந்தன்னில்
எய்தசுமரி யென்றேமுன் னியம்பின ரறிவின்மிக்கோர்"

-தன்வந்திரி வைத்தியகாவியம்

- Burning sensation in urethra due to azhal kutram
- Burning micturition
- Formation of stone that appear like Semicarpus anacardium seeds

சுக்கில அச்மரி (SUKKILA ACHMARI)

"சுக்கிலம் வருங்காலத்தில் தம்பித்தாற் சுக்கிலந்தான்
மிக்கக்கல் லாகிவெதும்பி விதனமாய் நீர்விடாமற்
சிக்கிநீர் விழாமலங்கே மணல்விழும் வெளுக்கும்தேகம்
மிக்குணஞ் சுக்கிலாசு மரியசாத்திய மென்றோரே"

-தன்வந்திரி வைத்தியகாவியம்

- Suppressions of semen during ejaculation, develops on to stones and obstruction in the flow of urine
- Sand like gravels are expelled
- Pallor of the body
- This is curable

Classification of disease in Siddhar Aruvai Maruthuvam:

1. Vali kalladaippu
2. Azhal kalladaippu
3. Iyya kalladaippu
4. Venneer kalladaippu

Classification in Jeevaratchamirtham and Anubava vaithya devaragasium:

Five types

1. Vatha achmari
2. Pitha achmari
3. Kabha achmari
4. Sukkila achmari
5. Swargara achmari

Classification of disease in North books:

1. Vali kalladaippu
2. Azhal kalladaippu
3. Iyya kalladaippu
4. Sukkira kalladaippu
5. Sarkkarak kalladaippu

-Noi naadal and Noi muthal naadal part I

MUKKUTRA VERUPADUKAL (PATHOLOGY)

The imbalance in one's diet and fluid intake increases the Azhal kutram. This raised azhal kutram dries up the body fluid and urine resulting in concentration of salts; this further affects the keezh nokku kaal. One of the functions of the keezh nokku kaal is to excrete urine. So when this keezh nokku kaal is affected the urine will be obstructed within urinary tract. This favors the deposition of urinary salts to develop into calculi anywhere in the kidney or urinary tract.

வாயு புகுந்து மலத்தோட பானத்தைத்
தேயு கூட்டித் திரட்டிச் சுருக்கும்
தேயும் மலம் வரில் சுருக்கி முன்னே
நின்றேயு முனை போல பானனிற்கும்

- சித்த மருத்துவாங்கச் சுருக்கம்

நோய் கணிப்புமுறை (DIAGNOSIS)

In piniyarium muraigal the following principles are followed in siddha system. There are,

1. Poriyal therthal
2. Pulanal arithal
3. Vinathal

The Maruthuvar (physician) should observe the patient, palpate and interrogate the patient thouroughly. This is stressed also understood by this maxim.

“Eyes first and most, Hands next and little, mouth last and never”

PORIYAL THERTHAL & PULANAL ARITHAL

Poriyal therthal or understanding by the five organs of perception.

Pulanal arithal or understanding by the sense objects. There are,

1. Mei - Ooru (somatic sense)
2. Vaai -Suvai (taste)
3. Kan - Oli (vision)
4. Mooku- Natram (smell)
5. Sevi - Osai (sound)

VINATHAL

An effective history taking helps one to diagnosis properly. By vinathal the physician should ask the patients native place, mode of living, food habits, complaints and duration of illness etc. if the patient is deaf or dump or if the patient is a child, the particulars should be obtained from his relatives or parents.

Poriyal therthal, pulanal arithal and vinathal are applied through eight special tools of investigation that is envagai thervugal.

ENVAGAI THERVUGAL

"நாடிப்பரிசம் நாநிறம் மொழிவிழி
மலம் மூத்திரமிவை மருத்துவராயுதம்"

-தேரையர்

"மெய்க்குறிநிறம் தொனி விழிநா இருமலம்
கைக்குறி" **-தேரையர்**

கல்லடைப்பு நாடி நடை

Aggravation of vali naadi produces symptoms of **Kalladaippu.** This is emphasized in Agathiar Naadi, Sathaga Naadi and Rathina churukka Naadi.

"அறைந்தோம் வாதரோகியுடல்
அடிக்கண் முகமும் பலமலமும்
நிறைந்த விழியில் நீர்வடியும்
நீண்ட நாவு கறுத்திடவும்
நிறைந்த முள்ளாய் தானிருக்குஞ்
சிறுநீர் பொருமி கருத்து வரும்
உறைந்த நீருங்கரு கருத்து
முறையாய் ரோகமு முண்டாய்"

- அகத்தியர் நாடி

"வாதமெனும் நாடியது தோன்றில்
சீதமந்தமொடு வயிறுபொருமல் திரட்சிவாயு
சீதமுருங் கிராணி மகோதரம் நீராமை
திரள்வாயு சூலை வலிகடுப்புத்தீரை
நீதமுருங் கிருமிகுன்மம் அண்ட வாதம்
நிலையுநீர்க் கிரிச்சரங்கள் தந்து மேகம்"

-சதக நாடி

"மேவிய வாதஞ்செய்யும் குணத்தை விரும்பிக்கேளு
தாவிய வயிருமந்தஞ் சந்துக்கால் பொருத்துநோவாம்
சேவிய தாதுநாசச் சிறுத்துடன் சிறுநீர் வீழும்
காவியங் கண்ணீனாளே மலமது கருக்கிகாணும்"

- இரத்தினச் சுருக்கநாடி

Aggravation of Azhal naadi produces symptoms of **Kalladaippu,**

"ஏவலாய் குழலாய் பித்தசெய்குணம் விளம்பக்கேளாய்
கோலவேல் விழிசிவந்து குளிர்ந்திருக்கு மல்லால்
சீலவே நீர்கருத்து நொந்து சுருக்கென வந்துவிழும்
ஞாலமே கிறுகிறென்று நாவுலர்ந்திருக்குந்தானே"
"பித்தரோகி பெருமுடல் சூடாகும்
நித்தமா முகம்நேர் விழி நாவுபல்
முத்த நீரு முயர்ந்த சிவப்பாகும்
சுத்த மஞ்சளாய்த் தோன்றிடக் கண்டிடே"

- இரத்தினச் சுருக்கநாடி

Derangement of Valiazhal naadi produces symptoms of **Kalladaippu,**

"பொருளான வாதத்தில் பித்தஞ் சேர்ந்து
பொருந்து குணங்களா முஷ்ணவாயு சத்தி
செரியாமை புளித்தேப்பம் பொருமல் நீரிற்
சிவப்புமலம் பிடித்தலுருந் தாது நட்டம்"

- சதக நாடி

ஸ்பரிசம் (Touch)

By sparism the temperature of skin (thatpam - cold or veppam - heat), sweating, dryness, smoothness, roughness, hard patches, swelling, abnormal growth of organs and tenderness can be felt.

In kalladaippu patients tenderness over the lower abdomen, lumbar region, renal angle and swelling can be felt (may be due to hydronephrosis). The patient's temperature is also increased in lower abdomen and sweating all over the body at the time of colic.

நா (Tongue)

By the examination of the tongue its colour, size, coating, moisture, ulcer, fissure, crust, movement and condition of teeth and gums can be examined. In kalladaippu if there were constipation, the tongue would seem to be coated. Loss of taste in Karuneer kalladaippu.

"கருநீர்க்கல்லின் வளி சினந்தெழுந்து
சுவைகெடல் வெளிறு மறுப்பு நீர்வேட்கை"

- நோய் விளக்கம்

நிறம் (Colour)

Colour of the skin, conjunctiva, tongue, nail bed and hair etc.

- Vali udal - Black colour
- Azhal udal- Yellow or red colour
- Iyya udal - White colour

மொழி (Speech)

By examining mozhi (speech), characters, hoarseness, slurring speech and various disorders of speech such as dysarthria can be noted. In kalladaippu there is low pitch voice due to agonizing pain in lower abdomen and burning sensation.

விழி (Eye)

Examine the colours of eye like reddish or yellowish discoloration and characters like dryness and lacrimation. Tiredness and redness due to pain is observed in patients with renal colic. In addition one has to be examine the patients acquit; there may be pallor of eyes due to gross haematuria.

மலம் (Stool)

By examining Malam, its nature, colour, quantity and presence of blood or pus can be noted.

நீர்க்குறி (Urine examination)

Urine examination is good diagnosis method compare to other Envagai thervugal. Theraiyar mentions below as

நீர்குறிச் சிறப்பு

"தர்க்கசாத் திரிக ளானோர்
தங்களிற் றேர்ந்து நாடி
வர்க்கமாம் நாடி தன்னில்
வருவது மயக்க மென்றே
உற்றநீர்ப் பரீக்ஷி யாய்ந்தே
யுரைத்தன ரிதற்கு நேராய்
மற்றொரு விதிநூ லில்லை
மருத்துவக் கலைவல்லோர்க்கே"

- சித்த மருத்துவாங்கச் சுருக்கம்

Siruneer should be collected in early morning, patient should be eating six tastes of food with regular time and well sleeping overnight, urine should be examine within 3 3/4hrs. This is quoted as

"அருந்துமா றிரதமும் அவிரோ தமதாய்
அஃகல் அலர்தல் அகாலவூண் தவிர்ந்தழற்
குற்றள வருந்தி உறங்கி வைகறை
ஆடிக் கலசத் தாவியே காதுபெய்
தொருமுகூர்த் தக்கலைக் குட்படு நீரின்
நிறக்குறி நெய்க்குறி நிருமித்தல் கடனே"

- சித்த மருத்துவாங்கச் சுருக்கம்

சிறுநீரின் பொதுக் குணம்

"வந்தநீர்க் கரியெடை மணம் நுரை எஞ்சலென்
றைந்திய லுளவை யறைகுது முறையே"

- சித்த மருத்துவாங்கச் சுருக்கம்

1. நிறம்(Colour)
2. எடை(Specific gravity)
3. நாற்றம்(Smell)
4. நுரை(Froth)
5. எஞ்சல்(Deposits)

Above the five parameters by which each urine sample should be examined.

நிறம் (COLOUR)

"பீதம் செம்மைபைங் கருமை வெண்மையென்
றோதைங் கொழுமையை யொத்துகு நீரே"

- சித்த மருத்துவாங்கச் சுருக்கம்

1. Yellow
2. Red
3. Green
4. Black
5. White

Urine may be any colour mentioned above.

கல்லடைப்பு நீரின் குணம் (Colour Indicating Renal stones)

The urine colour would look like flesh washing water; this is indicated in kidney diseases.

"தீப்புலால் கழுநீர்ச் செயலெனிற் குண்டிக்
காய்த்துர்ப் பலத்தால் கதித்த நீராமத்
துர்ப்பலக் கபமும் சோரியும் கொதிப்புறப்
பற்பக லாகப் பையப் பதிந்ததே"

- சித்த மருத்துவாங்கச் சுருக்கம்

எடை (SPECIFIC GRAVITY)

No thickness in urine is considered to be healthy.

"மிகத்தடிப் பும்மிகத் தேறலும் இன்றெனில்
சுகத்தைத் தரும்மெய்ச் சுபாவநீர் நன்றே"

- சித்த மருத்துவாங்கச் சுருக்கம்

நுரை (FROTH)

"பந்தமெய்ப் பசையிள கப்படும் பருவத்
தந்தர்ப் பூதமாய் அனிலமூத் திரத்தில்
சம்பந்தப் படும் ததிநுரைப் புனலே"

- சித்த மருத்துவாங்கச் சுருக்கம்

Urine may be frothy in nature. If it is reduced, vali, azhal and iyyam are said to be deranged.

நாற்றம் (SMELL)

மணவிலக்கணம்

"ஓதமணத் தோடவ் வோதமொத் திறங்கும்
சீதளங் கம்மிய தேகிக ளுக்கே"

- **சித்த மருத்துவாங்கச் சுருக்கம்**

"காதிணில் சீழுங் கலந்திழி மணமுறின்
கருப்ப நாபிகளு ளுங் காம நாளத்துளும்
விரணமுன் டின்றேல் எய்துகல் மறியல
திருத்தலே திண்ண மெனமனத் துன்னே".

- சித்த மருத்துவாங்கச் சுருக்கம்

The presence of pus of an obnoxious odour suggests infections and ulcer of the genitalia. This also occurs in renal calculi.

எஞ்சல் (DEPOSIT)

If urine excretion look like curd water, milk and sand like deposits in urine indicate stones in kidney. This is mentioned as follows,

"நார்த்ததி நீர்பால் போல
நவையுற்றங் கிழியு மானால்
மாரற்ப முற்ற நீரி
லடிமண்டிக் கிடந்த தானால்
பாரிந்த மெழுகு மாங்காய்
பற்றிய கல்லி னாலே
சீருற்ற செய்கை யென்று
தெரிவுறச் செப்ப லாமே"

- சித்த மருத்துவாங்கச் சுருக்கம்

நெய்க்குறி

The urine is kept in a bowl; a drop of oil gently with rod is dropped at the centre of urine bowl without any shake. It should be ensure that the sunlight falls on it, but is not disturbed by the wind. A keep observation of the oil drop suggests the condition of the patient.

நிறக்குறிக் குரத்த நிருமாண நீரிற்
சிறக்க வெண்ணெய்யோர் சிறுதுளி நடுவிடுத்
தென்றுறத் திறந்தொலி ஏகாதமைத்ததி
னின்றதிவலை போம் நெறிவிழியறிவும்
சென்றது புகலுஞ் செய்தியை யுணரே

- சித்த மருத்துவாங்கச் சுருக்கம்

If oil spreads like the shape of snake it indicates Vali neer, a ring indicates Azhal neer, if it stands like a pearl it indicates Iyya neer and sinks in urine indicates Mukkutram.

"அரவென நீண்டிடின் அஃதே வாதம்"
"ஆழிபோற் பரவின் அஃதே பித்தம்"
"முத்தொத்து நிற்கின் மொழிவதென் கபமே"

- சித்த மருத்துவாங்கச் சுருக்கம்

தீரும் தீராதவை (PROGNOSIS)

"சூட்டிட்ட சாத்தியத்தைச் சொல்லக் கேளாய்
சுளுக்காகும் வாதத்தின் கல்ல டைப்பு
பூட்டிட்ட பித்தத்தின் கல்ல டைப்பு
புகழான சிலேட்டுமத்தின் கல்ல டைப்பு
மூட்டிட்ட இதுமூன்றும் சாத்திய மாகி
முனையான மருந்துகளிற் செம்மை யாகும்
தோட்டிட்ட தொந்தமாங் கல்ல டைப்புத்
தொடுசுறவே கொல்லுமிது சூட்சந் தானே"

- யூகி வைத்தியசிந்தாமணி.

According to Yougi mamunivar, vali, azhal and iyya kalladaippu are curable whereas mukkutra kalladaippu is incurable.

மருத்துவம் (LINE OF TREATMENT)

"வைத்தியச் செயல் வைத்தியமே"

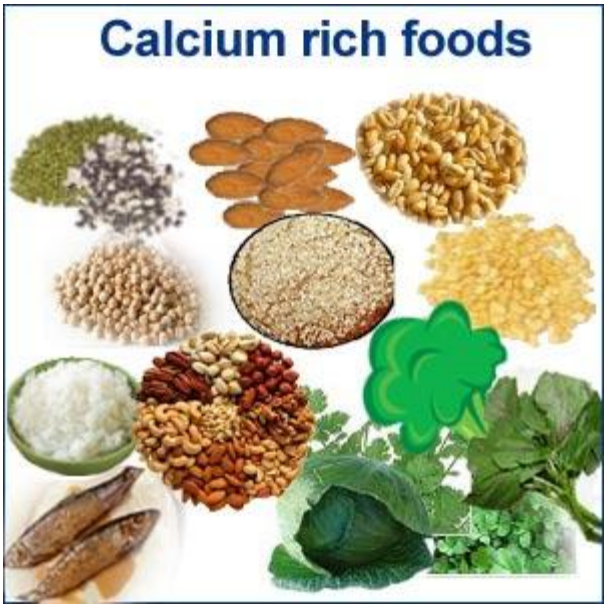
- திருமூலர் 800

The author of dissertation has selected trial drug **Karpoora Silasathu Parpam**, dose of 130mgs two times per day with Radish juice for 48 days.

In siddha system, treatment is not only for removable of disease but also the prevention and improving the body condition after removal of disease. This is said as kaappu, neekkam and thiraippu.

DIET

All patients are strictly advised to follow the following diet restriction. All the kalladaippu patients in this trial should avoid highly calcium and oxalate diet like,



OXALATE FOODS LIKE		CALCIUM FOODS LIKE	
• Spinach • Beans • Green pepper • Salt • lots of meat • soft drinks • cocoa • tea	• Cabbage • cauliflower • Tomato • peanuts • Cashew nuts • Almond • Grapes • strawberry	• milk • buttermilk • curd • butter • cheese	• panneer • milkgova • chocolates • fish • egg

According to Siddha Maruthuvam

சேர்க்கவேண்டியவை:

- நாள் ஒன்றுக்கு **2 முதல் 3 லிட்டர்** வரை தண்ணீர் அருந்த வேண்டும்.
- பார்லி அரிசிக் கஞ்சி, நன்னாரி வேர்க்குடிநீர், இளநீர் ஆகியவை சேர்க்க வேண்டும்.

காய்கள்: முள்ளங்கி, அவரை, வெண்டை, சுரைக்காய், வெண்பூசணி, கீரைத்தண்டு, வாழைத்தண்டு, கேரட், பீர்க்கங்காய்.

கீரைகள்: சிறுகீரை, தாளிக்கீரை, காசினிக்கீரை, புதினாக்கீரை, கருவேப்பிலை

பழங்கள்: தர்பூசணி, வெள்ளரிப்பிஞ்சு, அன்னாசிப்பழம், பப்பாளி, வாழைப்பழம், எலுமிச்சை

தானியங்கள்: கடலைப்பருப்பு. உளுந்து, பாசிப்பயறு, உலர்ந்த பட்டாணி

தவிர்க்க வேண்டியவை:

➢ தக்காளி	முட்டைக்கோசு	காளிபிளவர்
➢ மீன்	முட்டை	மாமிச உணவு
➢ காளான்கள்	கீரைகள்	முருங்கைக்காய்
➢ திராட்சை	ஸ்ட்ராபெர்ரி	சாக்லேட்
➢ மதுபானம்	காபி/டீ	சமையல்சோடா
➢ புகையிலை	புளி	வெற்றிலை/பாக்கு

- ➢ பதப்படுத்தப்பட்ட குளிர் பானங்கள்
- ➢ உப்பு நிறைந்த உணவுகள் மற்றும் நீர்
- ➢ பாலில் தயாரிக்கப்பட்ட உணவு வகைகள்
- ➢ பொரிக்கப்பட்ட மற்றும் மசாலா சேர்ந்த உணவு வகைகள்.

RENAL CALCULUS

ANATOMY OF THE KIDNEY

The Kidneys are a pair of the major excretory organ in the body. Each kidney is bean shaped situated on the posterior abdominal wall, on either side of the lumbar vertebral column. The left kidney is a little longer and narrower than the right kidney. Each kidney weighs about 120 to150gms. It is a compound tubular gland covered by a connective tissue capsule. There is a depression on the medial border of kidney called hilum, through which renal artery, renal veins, nerves and ureter pass. The components of kidney arranged in three layers.

1. Outer Cortex

This is dark and granular in appearance. It contains renal corpuscles and convoluted tubules. At intervals, cortical tissue penetrates medulla in this form of columns which are called renal columns or columns of Bertini.

2. Inner Medulla

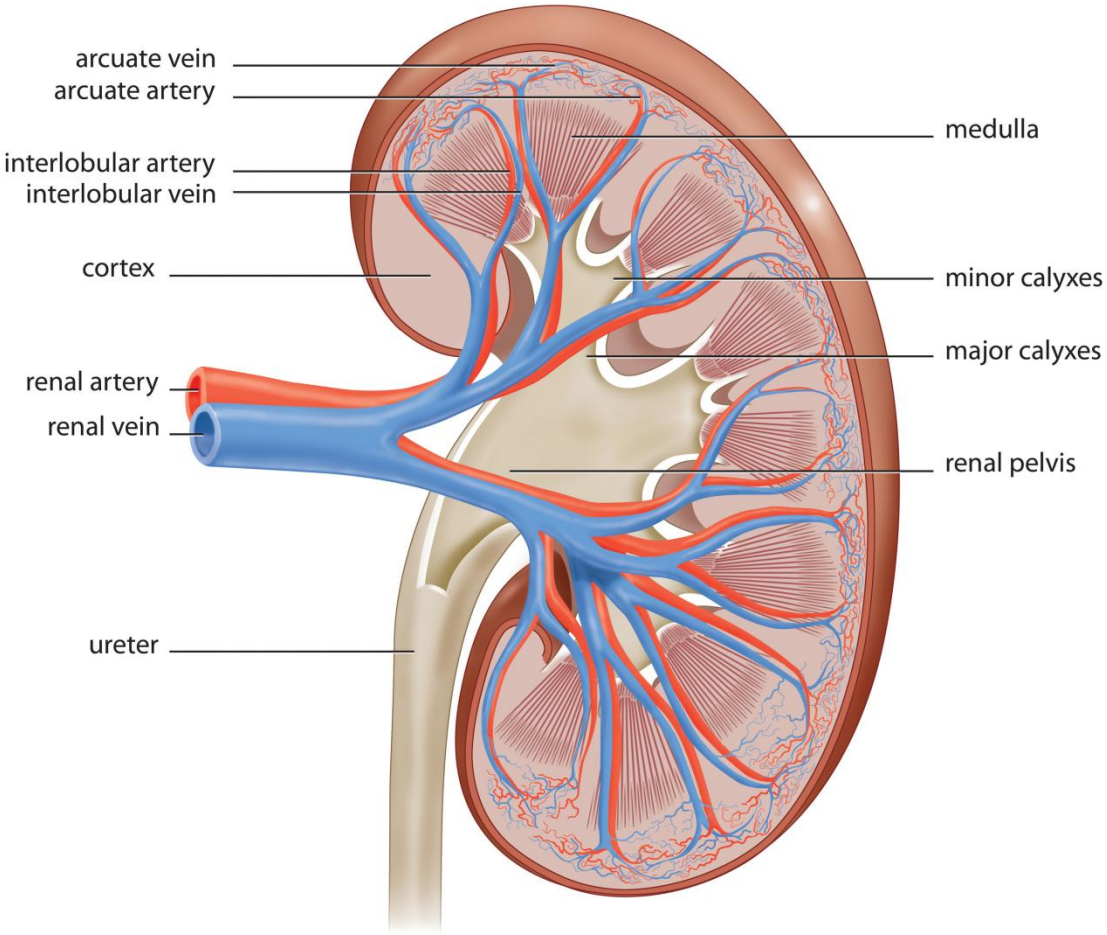
This gives radially striated appearance as it contains tubular and vascular structures. Medullary mass is divided into 8 to 18 medullary or malpighian pyramids. Broad base of each pyramid is in contact with cortex and the apex projects into minor calyx.

3. Renal Sinus

It consists of the following structures;

- Upper expanded part of ureter called renal pelvis.
- Subdivisions of pelvis - 2 or 3 major calyces and 8 minor calyces.
- Branches of nerves and arteries and tributaries of veins.
- Loose connective tissues and fat.

KIDNEY


arcuate vein
arcuate artery
interlobular artery
interlobular vein
cortex
renal artery
renal vein
ureter
medulla
minor calyxes
major calyxes
renal pelvis

NEPHRON

Nephron is defined as the Structural and functional unit of the kidney. Each kidney has a million nephrons. Each nephron is formed by two parts called renal corpuscle or malpighian corpuscle and renal tubule. Each nephron begins in cortex as a funnel like

dilatation called the Bowman's capsule, which encloses a tuft of capillaries, the glomerulus. The Bowman's capsule together with glomerulus is called the Renal corpuscle.

The renal tubule leaves the Bowman's capsules and becomes convoluted to form the proximal convoluted tubule (PCT). It then becomes straight and passes down the medulla as the descending limb of the loop of Henle, after varying distances before reaching the end of the papilla, it turns round in the form of U-shaped bend, forming loop of Henle, and passes upwards towards the cortex, parallel with its former course as the ascending limb of the loop of Henle.Each limb has an outer thick and inner thin portion.

Thick ascending limb approaches its own, glomerulus, and contact with the afferent and efferent arterioles to form the juxtaglomerular apparatus. It then becomes convoluted to form the distal convoluted tubule (DCT). The DCT then straightens out and joins by short connecting ducts to form the collecting tubules or collecting ducts (CD). They unit to form larger collecting ducts and descend parallel to loop of Henle and open in the papilla or the duct of Bellini in the renal pelvis. The length of the nephron varies from 4 to 6.5cms.

RENAL CIRCULATION

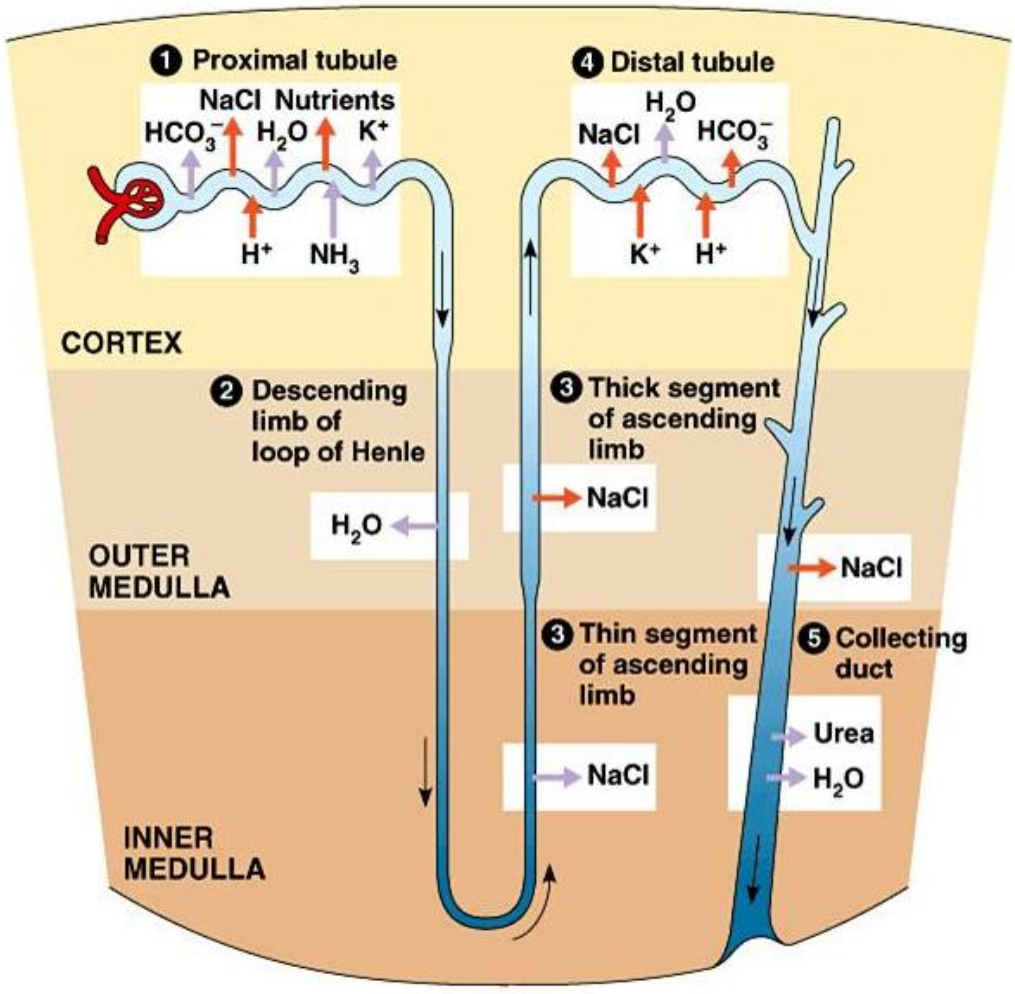
Renal artery arises from the abdominalaorta, enters the kidney through the hilus and divided into an anterior and a posterior branch, which gives rise to about five segmental arteries. The segmental artery divided into interlobar arteries, which pass outward in the medulla between the pyramids to reach boundary zone between the medulla and cortex. Here they turn to take a horizontal course uniting with adjacent arteries to form arterial arches called arcuate arteries. Several straight arteries arise from these arches and run radially outward through the cortex. These are called interlobular arteries. From each interlobular artery, numerous afferent arteries arise, and enter the Bowman's capsule forming glomerular capillary tuft. The afferent

arterioles divide into 4 to 5 large capillaries, which form the loop, and capillary loop unite to form the efferent arteriole, which leaves the Bowman's capsule.

The efferent arterioles give rise to renal portal system. The efferent arterioles form a second capillary network surrounding the tubular portion of the nephron; the capillaries of second set are called peritubular capillaries. Thus the renal circulation forms a portal system by the presence of two sets of capillaries – glomerular capillaries and peritubular capillaries.

The tubular portion of juxtamedullary nephrons are supplied by some specialized capillaries called vasa recta.Vasa recta arise directly from the efferent arteriole of the juxtamedullary nephrons. The peritubular capillaries and vasa recta drain into the venous system, which include the peritubular venules, interlobular veins, arcuate veins, interlobar veins, segmental vein and finally the renal vein. Renal vein leaves the kidney through the hilus and join the inferior vena cava.

NEPHRON



URINE FORMATION

Kidney excretes the unwanted substances along with water and urine includes metabolic end products and those substances, which are present in excessive quantities in the body, through urine. Normally about 1 to 1.5 liters of urine is formed every day. The urine formation includes the following three processes:

1. Glomerular filteration
2. Tubular reabsorption
3. Tubular secretion

GLOMERULAR FILTERATION

When blood passes through the glomerular capillaries the plasma is filtered into the Bowman's capsule. All the substances of plasma are filtered except plasma proteins. The filtered fluid is called glomerular filtrate. During filtration the substances passes through the three layer of filtrating membrane such as,

1. The glomerular capillary membrane
2. Basement membrane
3. Endothelium of visceral layer of Bowmen's capsule.

The glomerular filtration is called ultrafiltration because even the minute particles are filtered, but the plasma proteins are not filtered due to larger molecular size than the size of the slit pores present in the endothelium of capillaries. The composition of glomerular filtrate is similar to that of plasma except in the absence of plasma proteins.

GLOMERULAR FILTRATE RATE (GFR)

The total quantity of filtrate formed in all the nephron of both the kidneys in the given unit of time is called glomerular filtrate rate. The normal value of glomerular filtrate rate is 125ml/ minute or about 180liters/day.

FILTRATION FRACTION

The fraction of the renal plasma, which becomes the filtrate is called filtration fraction. It is the ratio between renal plasma flow and glomerular filtration rate. It is expressed in percentage, as follows,

$$\text{Filtration fraction} = \frac{\text{GFR} \times 100}{\text{Renal plasma flow}}$$

$$= \frac{125\text{ml/minute} \times 100}{650\text{ml/minute}}$$

$$= 19.2\%$$

The normal filtration fraction values from 15 to 20%

PRESSURES DETERMINING FILTRATION

Glomerular capillary pressure, colloidal osmotic pressure in the glomeruli and the hydrostatic pressure in the Bowman's capsule, which are determine the GFR. Among these pressures, the glomerular capillary pressure favors filtration the glomerular capillary pressure, is about 60mmHg and is the highest capillary pressure in the body. The colloidal osmotic pressure and hydrostatic pressure oppose the filtration. The colloidal osmotic pressure exerted by plasma protein in the glomeruli. The plasma proteins are not filtered through the glomerular capillaries, so increased concentration of proteins in glomerulus during filtration causes the development of colloidal osmotic pressure. It is about 25mmHg. Hydrostatic pressure in Bowman's capsule is exerted by the filtrate in Bowman's capsule during filtration. It is about 15mmHg.

NET FILTRATION PRESSURE

The balance between pressure favoring filtration and pressures opposing filtration is called net filtration pressure. It is very essential for the maintenance of GFR, so this is otherwise known as effective filtration pressure of essential filtration pressure.

The net filtration pressure = Glomerular capillary pressure – colloidal osmotic pressure + Hydrostatic pressure in Bowman's capsule

$$= 60 - (25+15)$$

$$= 20\text{mmHg}$$

The normal net filtration pressure is about 20mmHg and it varies between 15 to 20mmHg.

FILTRATION COEFFICIENT

It is the GFR per mmHg of net filtration pressure.

$$\text{The filtration coefficient} = \frac{125\text{ml}}{20\text{mmHg}}$$

$$= 6.25\text{ml/mmHg}$$

FACTORS REGULATING GFR

Following are the various factors, which regulate or affect the GFR

1. Renal blood flow

This is the most important factor necessary for glomerular filtration. GFR is directly proportional to renal blood flow. Normal blood flow to both the kidneys is 1300ml minute. The renal blood flow is controlled by autoregulation.

2. Tubulo glomerular feedback mechanism

This is the process in which the GFR is constantly regulated by means of feed back from renal tubule. The macula densa of juxtaglomerular apparatus is responsible for this. When the glomerular filtrate passes through the end portion of thick ascending segment of renal tubule, the macula densa detects the concentration of sodium chloride and accordingly alters the GFR. If the concentration of sodium chloride is more, macula densa causes constriction of afferent arteriole and filtration rate decreases, the constriction of afferent arteriole may be due to the secretion of thromboxane A2 from macula densa.

3. Glomerular capillary pressure

The GFR is directly proportional to glomerular capillary pressure. When glomerular capillary pressure is increased the GFR is also increased, in turn depends upon the renal blood flow and arterial blood pressure. Normal glomerular capillary pressure is 60mmHg.

4. Colloidal osmotic pressure

The GFR is inversely proportional to colloidal osmotic pressure exerted by protein. During dehydration or increased plasma protein level, colloidal osmotic pressure is more and GFR is reduced. Normal colloidal osmotic pressure is 25mmHg.

5. Hydrostatic pressure in Bowman's capsule

The GFR is inversely proportional to this. The hydrostatic pressure in Bowman's capsule is increased in conditions like obstruction of urethra and edema of kidney beneath renal capsule. Normally it is 15mmHg.

6. Constriction of afferent arteriole

This reduces the blood flow to the glomerular capillaries and this in turn reduces GFR.

7. Constriction of efferent arteriole

If the efferent arteriole is constricted initially there is an increase in GFR because of stagnation of blood in the capillaries.

8. Systemic arterial pressure

However, increase in mean arteriole pressure upto 180mmHg or reduction upto 60mmHg does not alter renal blood flow or GFR. This is due to autoregulatory mechanism. Variation in pressure above 180mmHg or below 60mmHg affects the renal blood flow and GFR because auto-regulating mechanism fails beyond this range.

9. Sympathetic stimulation

The mild or moderate stimulation of sympathetic nerves does not causes any significant change either in renal blood flow or in GFR. This is due to auto regulation. Strong sympathetic stimulation causes severe constriction of the blood vessels needs to increase filtration initially, but later decreases.however, if the stimulation is continued for more than 30 minutes, there is recovery of both renal blood flow and GFR. It is because of reduction in sympathetic neurotransmitter.

10. Surface area of capillary membrane

GFR is directly proportional to the surface area of the capillary membrane. If the glomerular capillary membrane is affected as in the case of some renal diseases, the surface area for filtration decreases. So, there is reduction in GFR.

11. Permeability of capillary membrane

GFR is directly proportional to the permeability of glomerular capillary membrane.

12. Contraction of glomerular mesangial cells

Contraction of these cells decreases surface area of capillaries resulting in reduction of GFR.

TUBULAR REABSORPTION

Tubular reabsorption is the process by which water and other substances are transported from renal tubules back to the blood. When the glomerular filtrate flows through the tubular portion of nephron, both quantitative and qualitative changes occur. The tubular epithelial cells reabsorb large quantity of water, electrolytes and other substances. The substances, which are reabsorbed, pass into the interstitial fluid of renal medulla, and from here, the substances move into the blood in peritubular capillaries. As the substances are taken back into the blood, the entire process is called tubular reabsorption.

Selective reabsorption

The tubular cells of kidney selectively reabsorb the substances present in the glomerular filtrate, according to the needs of the body. So the tubular reabsorption is called the selective absorption.

MECHANISM OF REABSORPTION

The mechanisms involved in tubular reabsorption are of two types

1. Active reabsorption
2. Passive reabsorption

1. Active Reabsorption

The movement of molecules is against the electrochemical gradient. This needs liberation of energy and the energy is derived from ATP. The substances reabsorbed actively from the renal tubule are sodium, calcium, potassium, phosphates, sulphates, bicarbonates, glucose, amino acids, ascorbic acid, uric acid and ketone bodies.

2. Passive Reabsorption

In this process, the movement of molecules is more along the electrochemical gradients. This process does not need energy; the substances reabsorbed by passive transport are chloride, urea and water.

Site of Reabsorption

Substances reabsorbed from proximal convoluted tubule are glucose, amino acids, sodium, potassium, calcium, bicarbonates, chlorides, phosphates, uric acid and water. The substances reabsorbed from loop of Henle are sodium and chloride. The substances reabsorbed from distal convoluted tubule are sodium, calcium, bicarbonate and water.

REGULATION OF TUBULAR REABSORPTION

Tubular Reabsorption is regulated by three factors.

1. Glomerulotubular balance
2. Hormonal factors
3. Nervous factors

GLOMERULOTUBULAR BALANCE

When GFR increases, the tubular load of solutes and water in the proximal convoluted tubule is increased. It is followed by increase in the reabsorption of solutes and water.

HORMONAL FACTORS

Several hormones are increases or decreases sodium reabsorption in structure of nephron.

NERVOUS FACTORS

Activation of sympathetic nervous system increases the tubular reabsorption from renal tubules indirectly by stimulation of renin from the juxtaglomerular cell.

THRESHOLD SUBSTANCES

Depending upon the degree of reabsorption, the various substances are classified into three categories.

1. High threshold substances

The food substances like glucose, amino acid, acetoacetate ions and vitamins are completely reabsorbed do not appear in urine under normal condition. These substances can appear in urine, only if their concentration in plasma is abnormally high or in renal diseases. So these substances are called high threshold substances.

2. Low threshold substances

The substances such as urea, uric acid and phosphate are reabsorbed to little extend. These substances appear in urine even under normal conditions. Such substances are known as low threshold substances.

3. Non- threshold substances

The metabolic end products like creatinine are not at all reabsorbed and are excreted in urine irrespective of their plasma level. These substances are called non-threshold substances.

MECHANISM OF REABSORPTION

TUBULAR SECRETION

Tubular secretion is the process by which the substances are transported from blood into renal tubules. Some substances secrete into the lumen from the peritubular capillaries through the tubular epithelial cells. These known as tubular secretion or tubular excretion.

1. Potassium is secreted actively by sodium – potassium pump in distal convoluted tubules and collecting ducts.
2. Ammonia is secreted in the proximal convoluted tubule.
3. Hydrogen ions are secreted in the proximal and distal convoluted tubules. Maximum hydrogen ion is secreted in proximal tubule.

Thus by the process of glomerular filtration, selective reabsorption and tubular secretion urine is formed in the nephron. It is also concentrated by counter current mechanism and anti- diuretic hormone. Finally it passes through the ureter into the urinary bladder and is stored there until it is voided out.

RENAL CALCULUS

Renal calculi are formed by deposits of polycrystalline aggregates composed of varied amounts of crystalloid and organic matrix. They can vary in size and may be found anywhere in the urinary tract from the kidney to the bladder. Urinary stones have afflicted humans for centuries; the first reported cases are of bladder and renal calculi found in Egyptian mummies dated 4800BC.

EPIDEMIOLOGY

INTRINSIC FACTORS

AGE AND SEX

The peak incidence of renal calculi occurs between 20 and 40 years of age. Males are affected 3 times as frequently as females. Testosterone may cause increased oxalate production in the liver (predisposing to calcium oxalate stones) and women have higher urinary citrate concentrations (citrate inhibits calcium oxalate stone formation). Renal Stones in childhood are usually predisposed to by urinary infections, a metabolic defect or an anatomical abnormality.

GENETIC

Approximately 25% of patients with renal stones have a family history of stone disease and it is possible that urolithiasis is a polygenic defect with partial penetrance. Several disorders that have a clear genetic basis do predispose to stone formation (renal tubular acidosis, cystinuria, xanthinuria).

EXTRINSIC FACTORS

GEOGRAPHY

The prevalence of renal calculi disease is highest among those living in mountainous regions, deserts and tropical areas. However the capability of individuals to carry their stone risk from area to area suggests that the major tendency to stone formation resides in the individual.

CLIMACTIC AND SEASONAL FACTORS

There is an association between stone formation and environmental temperature with stone formation being more common in the summer months.

This tendency may be due to relative dehydration and the subsequent production of concentrated, acidic urine. Alternatively some workers suggest the increased exposure to sunshine leads to increased urinary calcium excretion via the increased production of vitamin D leading to hypercalciuria.

Ureteric stones become more prevalent during the summer, the highest incidence occurring a month or so after peak summertime temperatures, presumably because of higher urinary concentration in the summer (encourages crystallization). Concentrated urine has a lower pH, encouraging cystine and uric acid stone formation.

OCCUPATION

Sedentary occupations predispose to stones compared with manual workers.The risk of calcium oxalate and uric acid stones formation in Astronauts because of hypercalciuria, hypocitraturia, decreased pH and lower urinary volumes.

WATER INTAKE

Low fluid intake (<1200ml/day) predisposes to stone formation. Increasing water hardness (high calcium content) may reduce risk of stone formation, by decreasing urinary oxalate.

DIET

The dietary intake of certain food and fluid substances that lead to a higher urinary excretion of substrates that form stones increases the incidence of calculi. A high intake f oxalates, calcium, phosphates and other elements often lead to an excess excretion of them in urine.

High intake of animal protein causes high urinary oxalate, low pH, low urinary citrate, and High salt intake causes hypercalciuria. However, a reduced calcium diet can increase the risks of further stone formation. It is therefore important that the diet is balanced and a relative excess or total abstinence of these constituents is avoided.

PHYSICAL CHEMISTRY

Urinary stones occur when offending salt crystallises in the urine and this occurs when the urine is supersaturated with the substance. Calcium and oxalate crystallisation can be modified by the presence of so-called urinary inhibitors i.e. citrate, magnesium.

The earliest phase of crystal formation is known as nucleation. Crystal nuclei usually form on the surfaces of epithelial cells or on other crystals. Crystal nuclei form into clumps a process known as aggregation. Citrate and magnesium not only inhibit crystallization but also inhibit aggregation.These include other inhibitors of crystallisation, and aggregation namely pyrophosphate, glyosaminoglycans, nephrocalcin and Tamm Horsfall proteins.

Crystal aggregation and retention are necessary to lead to stone formation and therefore anatomical abnormalities such as medullary sponge kidney, pelviureteric junction obstruction and calyceal diverticulae increase the risk of stone formation.

Bacterial infection is well known to predispose to stone formation by alteration of urinary PH, production of urease and perhaps by increasing urinary matrix production. Finally, abnormal intra and inter cellular calcium transport may lead to crystal retention in the tubules acting as foci for crystal aggregation, retention and eventual stone formation.

Urinary supersarturation

↓

Crystallization

Nucleation, growth, aggregation

Renal tubular injury → ↓

Crystal–cell interaction

↓

Crystal growth, crystal aggregation

↓

Stone formation

TYPES OF CALCULUS

Basically the renal stones can be divided into two major groups

I. Primary stones

II. Secondary stones.

(I) PRIMARY STONES

They appear in apparently healthy urinary tract without any antecedent inflammation.

(a) Calcium oxalate

(b) Uric acid calculi

(c) Cystine calculi

(d) Xanthine calculi

(e) Indigo calculi

(f) **Struvitecalculi**

(II) SECONDARY STONES

They are usually formed as the result of inflammation.

(a) Triple phosphate calculus

(b) Mixed stones

CALCIUM OXALATE STONES

Calcium oxalate stones are the most frequent renal stones containing 70–75% with Dumbbell shape. It develops as a multifactorial process in which an imbalance between crystallization - driving and - inhibiting forces plays a fundamental role. So are most certainly also sub epithelial calcifications (Randall's plaques), and it is of note that a large fraction of calcium oxalate containing stones also contain calcium phosphate.

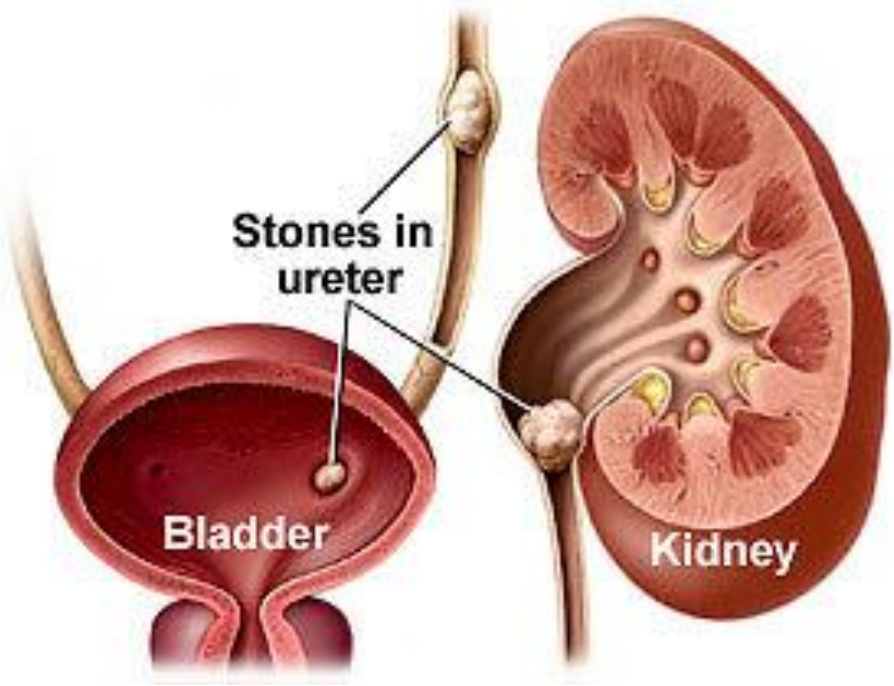
Calcium oxalate occurs in two different forms

Calcium oxalate monohydrate (COM) is compact and of brown or black color, varies in size and may have a spindle, oval, or dumbbell shape its formation is favored by high urinary oxalate concentrations.

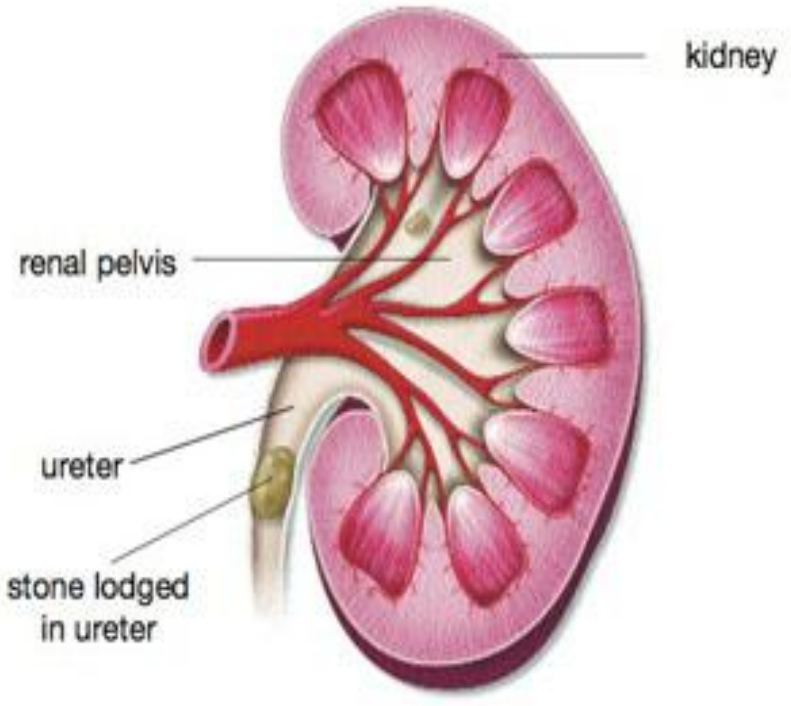
High concentrations of calcium and magnesium result in Calcium oxalate dehydrate (COD) stone formation. These crystals are varying in size, colourless squares whose corners are connected by intersecting lines. They can occur in urine of any pH. The recurrence risk is considered higher for COD than for COM stones.

Crystals of calcium oxalate form in supersaturated urine and then become anchored on the urothelium of the renal pelvis. Biochemical abnormalities that create supersaturation of the urine with respect to either calcium oxalate or calcium phosphate include hypercalciuria, hypocitraturia, hyperoxaluria, and hyperuricosuria.

SITE OF CALCULUS



URETERIC CALCULUS



PATHOPHYSIOLOGY OF STONE FORMATION

I. HYPERCALCIURIA

Hypercalciuria, or excessive urinary calcium excretion, occurs in about 5-10% of the population. It can be found in as many as 60% of all calcium stone patients. It is defined as urinary excretion of more than 250 mg of calcium per day in women or more than 275-300 mg of calcium per day in men while on a regular unrestricted diet. It can also be defined as the excretion of urinary calcium in excess of 4 mg/kg of body weight per day or as a urinary concentration of more than 200 mg of calcium per liter.

An alternate definition of hypercalciuria is daily urinary excretion of more than 3 mg of calcium per kilogram of body weight or more than 200 mg of calcium per day while on a restricted (400 mg calcium and 100 milliequivalent [mEq] sodium) diet.

The mechanism resulting in precipitation and growth of calcium crystals in the urinary tract are multiple and not fully understood hypercalciuria is recognized as an important and reversible risk factor in stone formation. Calcium homeostasis reflects the balance between absorption in the intestine, excretion by the kidney, and exchange from bone, the three processes being tightly regulated by endocrine mechanisms. Accordingly, the pathophysiology of hypercalciuria has been classified into (i) primary renal leak; (ii) imbalance between bone resorption and formation; and (iii) gut hyperabsorption. In turn, these primary defects trigger compensatory mechanisms primarily mediated by parathyroid hormone (PTH) and 1, 25-dihydroxy-vitamin D3 (1, 25$(OH)_2D_3$).

TYPES OF HYPERCALCIURIA

The most common types of clinically significant hypercalciuria are absorptive, renal leak, resorptive.

1. ABSORPTIVE HYPERCALCIURIA

Absorptive hypercalciuria is by far the most common cause of excessive urinary calcium. About 50% of all calcium stone formers have some form of absorptive hypercalciuria, which is caused by an increase in the normal gastrointestinal absorption of calcium, overly aggressive vitamin D supplementation, or excessive ingestion of calcium-containing foods (milk-alkali syndrome). Calcium

absorption occurs mainly in the duodenum and normally represents only about 20% of the ingested dietary calcium load.

Increased intestinal calcium absorption produces a corresponding increase in serum calcium levels. Typically, serum parathyroid hormone (PTH) is low or in the low-normal range in absorptive hypercalciuria, because the serum calcium level is generally high. Mild or moderate absorptive hypercalciuria can usually be controlled solely with dietary measures, but medical therapy is required in severe and resistant cases.

Absorptive hypercalciuria can be categorized into the following 3 types:

- **Type I** is relatively uncommon and is the most severe type of absorptive hypercalciuria. It is typically defined as the variant of absorptive hypercalciuria that is relatively unresponsive to dietary modifications, including severe dietary calcium restriction, but that normalizes urinary calcium excretion during periods of fasting.
- **Type II** is the most common variety of absorptive hypercalciuria and usually responds to moderate dietary calcium restriction.
- **Type III** also known as renal phosphate leak is a relatively uncommon cause of hypercalciuria. In this condition, the underlying etiology is a renal defect that causes excessive urinary phosphate excretion. This high urinary phosphate loss rapidly depletes the serum phosphate level, causing hypophosphatemia. The low serum phosphate increases the activation of vitamin D-3, which increases intestinal absorption of both phosphate and calcium. The unnecessary calcium absorbed is ultimately excreted in the urine, causing the hypercalciuria. Essentially, this is an absorptive vitamin D–dependent hypercalciuria due to inappropriate vitamin D-3 activation from hypophosphatemia. The hypophosphatemia is caused by the renal phosphate–losing defect.

2. RENAL LEAK HYPERCALCIURIA

Renal leak hypercalciuria is due to a specific defect in the kidneys that allows excessive obligatory urinary calcium excretion regardless of serum calcium levels, body stores, or calcium ingestion. The calcium/creatinine ratio is usually high (>0.20). The obligatory loss of serum calcium into the urine produces a mild hypocalcemia and secondary hyperparathyroidism, which is useful in diagnosing this condition. Renal leak hypercalciuria is far less common than absorptive hypercalciuria.

3. RESORPTIVE HYPERCALCIURIA (HYPERPARATHYROIDISM)

Resorptive hypercalciuria is due to the loss of calcium from the body's normal stores in the bony skeleton and is typically found in hyperparathyroidism. In this condition, calcium is released from bone in response to the increased activity of osteoclasts caused by excessive and inappropriate serum PTH levels. This causes significant hypercalcemia. Under normal conditions, PTH causes the kidney to limit calcium excretion, but, with the overwhelming serum calcium load produced with hyperparathyroidism, the kidneys are forced to excrete the extra calcium into the urine, causing the hypercalciuria.

4. IDIOPATHIC HYPERCALCIURIA

Idiopathic hypercalciuria is a familial disorder clinically associated with kidney stone production and reduced bone mineral content. An increase of intestinal Ca^{2+}absorption and a reduction of tubular Ca^{2+} reabsorption are involved in the development of the disorder.

II. HYPERCALCEMIA

Hypercalcemia is a condition in which the calcium level in blood is above normal.

CAUSES OF HYPERCALCAEMIA

1. Primary Hyperparathyroidism

The excess Para thyroid hormone triggers the release of too much calcium into the bloodstream. The bones may lose calcium, and too much calcium may be absorbed from food. The levels of calcium may increase in the urine, causing renal stones.

2. Malignancy associated hypercalcaemia

Malignancy is a common cause of elevated blood calcium. Up to 20% of individuals withcancer will develop hypercalcemia at some point in their disease.

Certain types of cancer, particularly lung cancer and breast cancer, as well as some cancers of the blood, such as multiple myeloma, increase the risk of hypercalcemia. Some malignant tumors produce a protein that acts like parathyroid hormone, stimulating the release of calcium from bones into blood. This is considered a paraneoplastic syndrome, the body's response to the presence of cancer or a substance the cancer produces. Spread of cancer (metastasis) to bones also increases the risk of hypercalcemia.

3. Familial hypocalciuric hypercalcemia

Familial hypocalciuric hypercalcemia is a condition that can cause hypercalcemia, a serum calcium level typically above 10.2mg/dL. there is usually a family history of hypercalcemia which is mild, a urine calcium to creatinine ratio <0.01, and urine calcium <200mg/day.

The perceived lack of calcium levels by the parathyroid leads to constitutively high levels of parathyroid hormone, and therefore hypercalcemia. Functionally, parathyroid hormone increases calcium resorption from the bone and increases phosphate excretion from the kidney which increases serum calcium and decreases serum phosphate.

4. Granulomatous diseases

Granulomatous diseases include tuberculosis, an infectious lung disease, and sarcoidosis, an inflammatory disease that usually begins in lungs. Elevated levels of calcitriol stimulate digestive tract to absorb more calcium, which raises the level of calcium in blood. Also, a rare genetic disorder known as familial hypocalciuric hypercalcemia causes an increase of calcium in blood because of faulty calcium receptors in body.

5. Hypervitaminosis

An excess of vitamin D causes abnormally high blood concentrations of calcium produce hypercalcemia, which can cause over calcification of bones, soft tissues, heart and kidney. It can also damage the kidney and produce renal stones.

6. Hyperthyroidism

Hypercalcemia occurs in up to 15 to 20 percent of patients with hyperthyroidism.T3 stimulatesbone resorption by the Activation of osteoclasts.

7.Glucocorticoids

Administration of glucocorticoids decreases bone resorption of calcium and intestinal calcium absorption and increases renal calcium excretion, leading to a substantial decrease in serum calcium concentration produce hypercalcemia.

8.Milk-alkali syndrome

Milk alkali syndrome also called Burnett's syndrome is characterized by hypercalcemia caused by repeated ingestion of calcium and absorbable alkali such as calcium carbonate, or milk andsodium bicarbonate. If untreated, milk-alkali syndrome may lead to metastatic calcification and renal failure.

9. Pheochromocytoma

The increased catecholamines probably stimulated the parathyroid glands to produce excessive parathyroid hormone, resulting in hypercalcemia.

10. Immobilization

Immobilization causes hypercalcemia in patients, whose underlying bone resorption is elevated, including children and adolescents, patients with Paget's disease, mild hypercalcemia of malignancy. These patients may diminish hypercalcemia and development of osteopenia, but resumption of weight-bearing is essential for resolution of hypercalcemia and hypercalciuria.

11. Dehydration

A common cause of mild or transient hypercalcemia is dehydration, because when there is less fluid in blood, calcium concentrations rise.

HYPEROXALURIA

Hyperoxaluria is a common abnormal finding in patients with calcium oxalate renal stones. Some degree of excessive urinary oxalate is found in 20-30% of all patients with recurrent calcium oxalate stones. It defined asurinary oxalate excretion that exceeds 40 mg/day.

Types of hyperoxaluria

Primary hyperoxaluria (types I and II), enteric hyperoxaluria, dietary hyperoxaluria, and idiopathic or mild hyperoxaluria

Primary hyperoxaluria

This rare form of hyperoxaluria is due exclusively to a genetic defect that causes a loss of specific enzymatic activity. With the normal metabolic pathway blocked, the alternative pathway that leads to oxalate production as an end-product of glycolate metabolism becomes extremely active, resulting in extremely high oxalate production.

- **Type I** it occurs in 1 per 120,000 live births and is transmitted as an autosomal recessive trait. In primary hyperoxaluria type I, the missing enzyme is alanine-glyoxylate aminotransferase (ie, the *AGT*gene) and is normally found only in the hepatic peroxisomes. This enzyme is necessary to detoxify glyoxylate. When alanine-glyoxylate aminotransferase is lacking, oxalate production soars.

Pyridoxine (vitamin B-6) is a cofactor in this chemical pathway, which normally converts glyoxylic acid ($C_2 H_2 O_3$) to glycine. When the pathway is blocked because of a deficiency or absence of this enzyme, the result is high levels of glycolic and oxalic acid, which readily convert to oxalate. Oxalate is then excreted in the urine, which leads to nephrocalcinosis and the eventual development of end-stage renal failure, usually in childhood.

- **Type II** the missing enzyme is D-glyceric dehydrogenase, which can be detected in leukocyte preparations. This deficiency promotes the conversion of glyoxylate to oxalate.

  The 2 types of primary hyperoxaluria result in approximately the same degree of hyperoxaluria. However, end-stage renal disease is slightly less common in patients with type II primary hyperoxaluria. Pyridoxine is generally not effective in patients with type II primary hyperoxaluria.

Enteric hyperoxaluria

Enteric hyperoxaluria accounts for approximately 5% of all cases of hyperoxaluria. It is due to a gastrointestinal problem usually associated with chronic diarrhoea. Malabsorption from any cause can result in enteric hyperoxaluria. The basic mechanism is competition for the available ingested calcium, the leading intestinal oxalate-binding agent. Most of the bile acids produced during digestion are reabsorbed in the proximal intestinal tract.

When this fails to occur, calcium and magnesium bind to these bile acids through saponification.This leaves very little free calcium available for absorption or binding with oxalate in the lower intestinal tract. Without the calcium necessary to adequately bind oxalate in the intestinal tract, additional oxalate is absorbed and then excreted in the urinary tract.

Increased intestinal membrane oxalate transport and absorption may also occur through direct exposure of the intestinal lining to excess bile salts and fatty acids, which increase the oxalate permeability of the colonic mucosa.

Hyperoxaluria has been reported to be the most common urinary metabolic abnormality in patients with stones who have undergone bariatric surgery.Oxalate absorption by the colon has been shown to increase up to 300-fold following small-bowel bypass surgery; therefore, these individuals must be screened for enteric hyperoxaluria. In some severe cases, the bypass may need to be reversed to control the hyperoxaluria. The recent proliferation of bariatric surgery cases may result in an increased prevalence of enteric hyperoxaluria in the future.

Dietary Hyperoxaluria

Dietary oxalate was thought to play a relatively minor role in hyperoxaluria and to account for only 10-20% of the total urinary oxalate produced. Recent evidence suggests that dietary oxalate plays a much more important role and may be responsible for 50% of the total urinary oxalate.

HYPERURICOSURIA

15-20% of patients with calcium stones have hyperuricosuria.80 and 90% of persons with hyperuricosuric calcium nephrolithiasis are men. It defined as urinary uric acid levels that exceed 800 mg/day in men and 750 mg/day in women. The most common cause of hyperuricosuria is increased dietary purine intake, but many other hereditary or acquired factors eg, gout may result in this condition. Hyperuricosuria may develop uric acid or calcium oxalate stones due to supersaturation of urine with monosodium urate. It may initiate calcium oxalate stone formation by the induction of heterogeneous nucleation or by absorption of certain inhibitors. Patients with calcium oxalate stones have greater urinary pH than Patients with mixed uric acid and oxalate stones.

HYPOCITRATURIA

Hypocitraturia generally defined as urinary citrate excretion less than 320 mg (1.67 mmol) per day for adults is a common metabolic abnormality in stone formers, occurring in 20% to 60%. It is more common in premenopausal women with stone disease than in postmenopausal stone-forming women.

Citrate is a known inhibitor of stone formation, working through a variety of mechanisms. In the renal tubule citrate complexes with calcium, increasing its solubility and reducing the concentration of free calcium in the urine. This calcium-

citrate complex limits calcium supersaturation and prevents nucleation of both calcium oxalate and calcium phosphate, at least partly through interactions with Tamm-Horsfall protein.The excretion of citrate in the urine is a function of filtration, reabsorption, peritubular transport, and synthesis by the renal tubular cell. Acidosis plays the important etiologic factor in hypocitraturiaPatients with chronic diarrhoea and inflammatory bowel disease frequently has hypocitraturia due to bicarbonate loss from the intestinal tract. Thiazide owing to hypokalemia with resultant intracellular acidosis may induce hypocitraturia.A diet rich in animal protein and strenuous physical exercisemay produce hypocitraturia.UTI with bacteria that degrade citrate lowers urinary citrate levels.

HYPOMAGNESURIA

The most common cause of hypomagnesuria is chronic diarrhoeal syndrome and inflammatory bowel disease. Many of the patients with hypomagnesuria also have hypocitraturia.

SEX HORMONES AND RENAL STONES

In males, Testosterone appears to promote stone formation by suppressing osteopontin expression in the kidneys and increasing urinary oxalate excretion.

Menopause and postmenopausal hormone (PMH) are associated with an increase in urinary calcium excretion, which may increase the risk for calcium-containing stone formation in postmenopausal women compared with premenopausal women. Estrogen deficiency increases the sensitivity of bone to parathyroid hormone, leading to a net increase in bone resorption and increased urinary calcium excretion.

CALCIUM PHOSPHATE STONES

Calcium phosphate stones constitute up to 10% of renal stones. Calcium phosphate stones are less common than calcium oxalate with Elongate, narrow in shape. It precipitates in urine with an alkaline pH > 6.3. Calcium phosphate stones typically occur in patients with metabolic or hormonal disorders such as hyperparathyroidism and renal tubular acidosis.

CAUSES OF CALCIUM PHOSPHATE STONES

Patients with renal tubular acidosis, in addition to a high urinary pH, they also develop hypocitraturia because of the associated systemic acidosis, as well as hypercalciuria, probably due to effects of bone buffering of the acid load. All of these also favor calcium phosphate precipitation. Renal tubular acidosis significantly reduces urinary citrate as well as total urinary acid levels and can lead to stone formation, usually calcium phosphate.

Hypercalciuria appears to be the primary abnormality, with calcium-induced interstitial and tubular damage possibly responsible for the RTA. it is the initial condition with subsequent acidemia promoting stone formation both by increased calcium phosphate release from bone during bone buffering of retained acid and by direct reduction in the tubular reabsorption of these ions. The degree of hypercalciuria is roughly proportional to the severity of the acidemia. The persistently high urine pH, which favors the precipitation of calcium and reduced citrate excretion. Since acidemia enhances proximal citrate reabsorption. Urinary citrate is normally a potent inhibitor of calcium stone formation, both by forming a soluble complex with calcium and by inhibiting stone growth by agglomeration of calcium crystals.

URIC ACID STONE

About 5–10% of all stones are formed from uric acid stones, normally show as brownish-white colour.Itform from high concentrations of uric acid in acidic urine.They also may form in association with conditions that cause hyperuricosuria with or withouthyperuricemia. A diagnosis of uric acid stone is supported by the presence of radiolucent stone in the face of persistent urine acidity, in conjunction with the finding of uric acid crystals in fresh urine samples.

Pathophysiology

When the concentration of uric acid in urine exceeds its solubility at the urine pH, uric acid changes from a compound dissolved in solution to an insoluble precipitate. Urate stones are formed by general mechanisms are overproduction, increased tubular secretion, or decreased tubular reabsorption, decreased urinary water content, or increased hydrogen ion concentration.

Uric acid results as a relatively insoluble end-product of purine metabolism. The concentration of uric acid in plasma depends on dietary ingestion, de novo purine synthesis, and uric acid elimination by the kidneys and intestine.

Causes

Urine pH from 6.0 to 5.0 has a greater influence on uric acid stone formation than the 24-hour urine excretion of uric acid. Uric acid stone formation is more determined by pH than by urine volume or urine uric acid concentrations. The normal range for uric acid excretion 500 to 600 mg per 24 hour, and excretions greater than 750 mg for women and 800 mg for men are considered elevated. Clinically significant crystal and stone formation require persistent hyperuricosuria, dehydration, or marked reduction in urine pH.

The cause of the low urine pH in uric acid stone-formers is not completely understood, but reductions in urine ammonium excretion appear to play an important role. Dehydration, which reduces urine volume also promotes a decline in urine pH and thereby increases urine uric acid concentration. Stone formers who over-excrete uric acid do so either as a result of excess dietary purine ingestion and metabolic conversion to uric acid, or to excess production of uric acid.

HYPERURICEMIA

Hyperuricemia, and particularly gouty arthritis, are far more common in men than in women. Only 5% of patients with gout are female, but uric acid levels increase in women after menopause.

Hyperuricemia is an excess of uric acid in the blood, above the level of 7 mg/dL.

Also important to blood uric acid levels are purines. Purine breaks down into uric acid. Increased levels of uric acid from excess purines may accumulate in tissues, and form crystals. This may cause high uric acid levels in the blood.

PATHOPHYSIOLOGY

Hyperuricemia is generally divided into 3 pathophysiologic categories, ie, uric acid underexcretion, uric acid overproduction, and combined causes.

Uric acid underexcretion

Most patients with gout are “underexcreters”, i.e. they have a defect in their renal handling of uric acid, as evidenced by a lower than normal ratio of uric acid clearance to glomerular filtration. Patients with gout excrete; on average 41% less uric acid than normal people for any have given plasma concentration of uric acid. This underexcretion is believed to be due mainly to decreased proximal tubular secretion.

Uric acid overproduction

The causes for hyperuricemia in overproducers may be either exogenous (diet rich in purines) or endogenous (increased purine nucleotide breakdown).

Combined of underexcretion and overproduction

The most common cause under this group is alcohol consumption, which results in accelerated hepatic breakdown of ATP and the generation of organic acids that compete with urate for tubular secretion.

STRUVITE STONES (INFECTION STONES)

About 9% of all stones are formed from Struvite stones with dirty-white colour. In worldwide the frequency of struvite calculi account for up to 30%.

Struvite kidney stones are called triple phosphate stones because of their combination of magnesium, ammonium and calcium/phosphate. They are found predominantly in women with chronic urinary infections and people suffering from abnormal urinary tracts. They are most often associated with infections of the urinary tract.

Struvite stones are more amorphous and spread out into the insides of the kidneys which give them their antler-like appearance.it can also form around calcium oxalate stones when the stone becomes infected. This is a potentially dangerous situation that can cause the stone to grow so big that it blocks the entire kidney.

CAUSES

Struvite stones are almost always caused by urinry trct infections due to bacteri that prodce certain enzymes. These enzymes raise the concentration of ammonia in the urine. Ammonia makes up the crystals that form stuvite stones. The stone promoting bacteria are usually Proteus, but may also induce Pseudomonas, Klebsiella, Providencia, Serratia and staphylococci. Women are twice as likely to have struvite stones as men.

PATHOPHYSIOLOGY

Normal urine is undersaturated with ammonium phosphate, and struvite stone formation occurs only when ammonia production is increased and the urine pH is elevated more than 8, to decrease the solubility of phosphate.

Urease breaks down urinary urea into ammonia and carbon dioxide. The ammonia that is formed takes up a hydrogen ion to become an ammonium ion, increasing the PH of the urine so that it becomes more alkaline. Because phosphate levels are increased in alkaline urine and because magnesium always is present in the urine, struvite stones forms.

CYSTINE STONES

Cystine stones are relatively rare, occurring in about 1% to 2% of persons who experience renal stone disease. Cystine stones are most common in young adults under age 40. Less than 3% of urinary tract stones are cystine stones.

Cystine crystals form hexagonal-shaped, that can be viewed upon microscopic analysis of the urine. The stones may be pink or yellow in colour, but later they turn to greenish due to exposure to air. Stones may be present in single, multiple, or large staghorn configurations. Pure cystine stones are not easily visible on plain x-rays due to their sulfur content.

CYSTINURIA

Cystine kidney stones are due to cystinuria, a genetic disorder of the transport of an amino acid called cystine that results in an excess of cystine in the urine and the formation of cystine stones.

Cystinuria is characterized by the inadequate reabsorption of cystine in the proximal convoluted tubules after the filtering of the amino acids by the kidney's glomeruli, thus resulting in an excessive concentration of this amino acid in the urine. Cystine may precipitate out of the urine, if the urine is neutral or acidic, and form crystals or stones in the kidneys, ureters, or bladder.

XANTHINE STONES

These stones are extremely uncommon and usually occur as a result of a rare genetic disorder. Pure xanthine stones are radiolucent but patients with xanthinuria there may be a calcium salt mixture to render. These stones tend to be small, round or oval.

XANTHINURIA

Pathophysiology

The primary organs affected in xanthinuria are the kidney and, to a lesser extent, skeletal muscle and joints. Kidney complications are initiated by the formation of xanthine crystals in the tubules, leading to parenchymal deposition and/or radiolucent stone formation. Xanthine's high rate of renal clearance and low solubility in urine creates an environment in the urine favoring crystallization. Thus, patients with volume depletion who have xanthinuria are at particular risk of forming xanthine crystals.

INVESTIGATION

BLOOD TEST

Createnine, Blood Urea, Uric acid, calcium, phosphorus, Electrolytes and complete blood count.

URINE ROUTINE

- Crystals in urine.
- Blood cells in urine
- Pus cells in urine.

URINE CULTURE COLONY COUNT & SENSITIVITY TEST

24-HOUR URINE TEST

Urine is collected during a 24-hour period and analyzed for calcium, citrate, uric acid, magnesium, phosphate, sodium, oxalate, pH (acid level), and total volume.

STONE ANALYSIS OF THE RETRIEVED CALCULUS

IMAGING TESTS

- X-Ray KUB
- Ultrasonograpy of kidney, ureter, and bladder
- Intravenous pyelogram (IVP),
- Retrograde pyelogram,
- Computed tomography (CT) scan.

PROPERTIES OF THE TRIAL DRUG

கற்பூரச் சிலாசத்து - ASPHALT

ACTION: Diuretic, Lithotriptic, Astringent, Styptic, Nutrient.

பொதுக்குணம்:

கல்லடைப்பு மேகங் கனதூலம் வித்திரதி
சொல்லடைக்கு நீரருகற் சோணிதக்கான் - மெல்லிடையார்க்
கில்லகச்சத் தில்லையெனு மிந்திரிய நட்டமுமாங்
கல்லகச்சத் தில்லையெனுங் கால்.

INDICATION: Renal calculus, gonerrhoea, ascitis, Burning micturition, oligospermia.

Ref: Gunapadam Thathu- Jeeva Vagupu, 2nd, 3rd edition, p.no:529, 530.

KARPOORA SILASATHTHU

(Crystallised foliated gypsum)

Shilajit is known to give the physical power and actually reducing the apparent age of a person. Shila means Rock and shilajit means Rock Borne or Rock like.

Karpoora silasaththu, which is combination of alum and iron available in nature and plenty in Nepal. It is taken out from the earth as thick sheets.

Ref: siddha material medica, page no: 404

Constituents:

Silajit contains an oil which when distilled is known as ichthyol. Benzoic acid and benzoates are present in large quantities. It contains,

- Urea - 65p.c.
- Water - 8.85p.c.
- Organic matter - 56.20p.c.
- Mineral matter - 34.95p.c. containing
    - Nitrogen -1.03p.c.
    - Lime - 7.80p.c.
    - Potash - 9.07p.c.
    - Phosphoric acid - 0.16p.c.
    - Silica - 1.35p.c.

Uses:

- Silajit is specially employed in genito-urinary diseases.
- It is used in gallstones, renal and bladder calculi, anuria, jaundice, enlarged spleen etc.
- It diminishes phosphaturia and is useful in phosphatic calculus.

Ref: Indian Materia Medica Vol – 2

BEFORE PURIFICATION

PURIFY WITH TENDER COCONUT

AFTER PURIFICATION

வெங்காரம் - SODIUM BIBORATE

வேறுபெயர்: பொரிகாரம், காரம், உருக்கினம், உருக்குமித்திரன், டங்கணம், தூமத்தையடக்கி

Taste: Sweet with Astringent

Potency: Veppam (hot) (வெங்காரம் வெய்தெனினும் நோய் தீர்க்கும்)

Action: Diuretic, Lithotriptic, Refrigerant, Emmenogouge,

பொதுகுணம்:

சொறிபுடையெண் குன்மநமை சோரி யாசம்
பறிகிரகணி கல் கல்லானம் பன்னோய்- நெறியைத்
தடங்கணங்க பங்கிருமி சர்ப்பவிடஞ் சந்நி
யிடங்கணங்க லக்கிற்போ மெண்.

Indication: renal calculus, all types of ulcers, bleeding piles, Burning micturition, Delirium, Itching, Dysentery.

Ref: Gunapadam Thathu- Jeeva Vagupu, 2nd, 3rd edition, p.no: 434, 435.

CHARACTERS:

Borax: Na2B4O7 10H2O; Na2O-16.2%, B2O3-36.6%, H2O-47.2%,
specific gravity: 1.7

Borax, also known as a hydrated sodium tetraborate, is one of the most important ofboron compound, a mineral, and a salt of boric acid. It is a soft and light crystalline substance. The colour is greyish white. Exposed to the air it becomes opaque or dirty white.

Ref: The wealth of India raw materials volume 2: B

USES:

- A mixture of powdered borax and honey is used externally for urethritis to the urethra and soreness of mouth.
- Borax is given internally in doses varying from 10- 30 grains (650mgs-2gm) in acidity of the stomach, amenorrhoea, menorrhagia, and puerperal convulsions.

Ref: Indian Materia Medica Vol – 2

BEFORE PURIFICATION

AFTER PURIFICATION

பருத்தி - Gossypium herbaceum, Linn.

வேறுபெயர்: ஆச்சாதநபலை, பரி, உத்திரி, காற்பாசம், பன்னல்

Part used: leaves, flower, tender, seed, bark, root bark

Taste: Sweet, Astringent
Potency: Veppam (hot)
Division: pungent
Action:

Leaves, Flower: Astringent, Nutrient

Seed: Laxative, Expectorant.

Bark, Root bark: Diuretic, Emmenogouge.

பொதுகுணம்:

பருத்தியிலை மொக்கிரண்டைப் பாலிலரைத் துண்ண
வருத்துகின்ற மேகமெல்லாம் மாறும் - பருத்த
விரத்தபித்தத் தோடு விரணவீக் கம்போம்
அரத்தவிதழ் மாதே! யறை.

Indication: Leucorrhoea, Hypertension, Wound, Swelling.

Ref: அகத்தியர் குணவாகடம்

BOTANICAL ASPECT:

At the central institute for cotton research, Nagpur, over 6000 accessions of germplasm in all the four cultivated species og gossypium have been assembled and evaluated. These include 390 in g.herbaceum linn.

Ref: The wealth of India, Raw materials, vol: 3: D-I

Botanical name: Gossypium herbaceum, linn.

Family: Malvaceae

Habitate: A small syrub, 60cm to 2.5m in height

Local name: eng - Indian cotton plant/ common cotton

Mal -parithi

San -karpasa

Hindi –kapas

PHYTOCHEMICALS:

- Gossypetin 8-0- rhamnoside
- Quercetin-3-0-glucoside
- Gossypol
- Strigol
- Betaine
- Choline
- Salicylic acid

Ref: Compendium of Indian Medicinal plants, vol: 4

Indian Material Medica, vol: 1

Medicinal uses:

- It is useful in vitiated conditions of vatha, pitha, stangury, burning sensation of the stomach.

  Ref: Indian Medicinal plants, vol: 3

- The leaves remove vatha, enrich the blood and increase the flow of urine.

  Ref: Indian Medicinal plants, vol: 1

துத்தி - Abutilon indicum

வேறுபெயர்: கக்கடி, கிக்கசி

Part used: leaves, flower, bark, root

Taste: Sweet

Potency: Thatpam (coolent)

Division: Sweet

Action: Diuretic, Nutrient, Laxative, Demulcent, Sedative.

பொதுகுணம்:

மூலநோய் கட்டி முளைபுழுப்புண் ணும்போகுஞ்
சாலவதக் கிக்கட்டத் தையலே!- மேலுமதை
எப்படியேனும் புகிச்ச எப்பிணியும் சாந்தமுறும்
இப்படியிற் றுத்தி இலையை.

Indication: piles, wound.

- The leave decoction with milk and sugar, it cures the Burning micturition.

Ref: அகத்தியர் குணவாகடம்

BOTANICAL ASPECT:

Botanical name: Abutilon indicum

Family: Malvaceae

Habitate: A syrub, 2-3 feet in height

Local name: **Mal -** thuththi

San - kanka-tila

Hindi -kanghi

PHYTOCHEMICALS

Leaves - Beta carotene

Flower - Mucilage, tannin, organic acids,

The essential oil contains,

- Alpha pinene
- Caryophyllene
- Caryophyllene – oxide
- Cineole
- Geraniol
- Geranylacetate
- Elemene
- Eudesmol
- Farnesol
- Borneol

Ref: The wealth of India, Raw materials, vol: 1: A-F

ROOTS- Asparagine, Gallic acid,

Ref: The Indian Material Medica vol: 1
Compendium of Indian Medicinal plants, vol: 4

MEDICINAL USES:

- The powder of seed have been taken, thus removes the burning sensation while passing urine.

Ref: Tamil -English dictionary, t.v.sambasivam pillai, vol: 4 part2

- Infusion of roots prescribed as a diuretic and demulcent in urethritis. Also used in relieving stangury and haematuria.
- Decoction of leaves is used as internally for stone in the bladder.

Ref: The Indian Material Medica vol: 1

- The milk of the plant cures urinary discharges.
- The bark is good in **stangury and haematuria.**

Ref: The Indian Medicinal plants, vol: 1

முள்ளங்கி - Raphanus sativus

Part used: leaves, seed, tuber.

Taste: pungent

Potency: Thatpam (coolent)

Division: Pungent

Action: Diuretic, Laxative, Stimulant, Aphrodisiac, Stomachic.

பொதுகுணம்:

வாதங் கரப்பான் வயிற்றெரிவு சூலைகுடல்
வாதங்கா சமையம் வன்தலைநோய்- மோதுநீர்க்
கோவைபன்னோய் பல்சிலந்தி குன்மமிரைப் புக்கடுப்புஞ்
சாவுமுள்ளங் கிக்கந்தத் தால்.

Indication: vatha diseases, eczema, ulcer, asthma.

Ref: அகத்தியர் குணவாகடம்

BOTANICAL ASPECT:

Habitate: An annular herb with short condensed stem.

Botanical name: Raphanus sativus, Linn.

Family **:** Brassicaceae

Local name: **Eng-** Radish

San- Moolika

Mal- mullanki

PHYTOCHEMICALS:

- **Roots-** amino acids, ornithine, citrulline, arginine, glutamic acid, aspartic acid

Ref: The Wealth of India, Raw Materials, vol: 5 R-Z

- Fresh vegetable contains 91.00 p.c. moisture
- **Completely dried material contain :**
    - Ether extract 4.00p.c.
    - Albuminoids 18.00 p.c.
    - Soluble carbohydrates 52.66 p.c.
    - Woody fibre 9.34 p.c.
    - Ash 16.00 p.c.

Ref: Indian Materia Medica Vol - 1

- **Seeds-** beta sitosterol, stearic acid

Ref: Compendium of Indian Medicinal plants, vol: 4

Medicinal uses:

- Radish leaves show broad spectrum antibiotic specific activity against streptococcus, pneumococcus, escherichia coli.

Ref: The Wealth of India, Raw Materials, vol: 5 R-Z

- Roots are used for urinary disease.

Ref: The Indian Medicinal plants, vol: 1

- Root juice is given for urinary complaints like dysuria and strangury.

Ref: Indian Materia Medica Vol - 1

கோழி முட்டை - EGG WHITE

வேறுபெயர்: சிற்றண்டம்

Action: Demulcent, Laxative, Nutrient.

பொதுகுணம்:

வாதபித்தஞ் சேர்ப்பிக்கும் வன்றோடம் புண்போக்குந்
தாதுவை மெத்த தழைப்பிக்கு- மோது
கபத்தை யடக்குங் கரப்பானுண்டாக்கு
மிபத்தையுறுங் கோழிமுட்டை யெண்.

Indication: vatha diseases, kaba diseases, wound.

- கோழி முட்டை வெள்ளைக்கரு நீரில் கரையும்.பதார்த்தங்களின் அழுக்கை எடுக்கவும் தேகத்தில் எரிச்சலைத் தணிக்கவும் வெள்ளைக்கரு உபயோகப்படுகிறது.

Ref: Gunapadam Thathu- Jeeva Vagup, 2nd, 3rd edition, p.no: 634& 635

- கோழி முட்டை குடல், மார்பு, சிறுநீர்ப்பை இவற்றை தூய்மையாக்கும். சிறுநீரகம், சிறுநீர்ப்பை இவற்றில் தோன்றும் இரணத்தைப் போக்கும்.

பார்வை நூல்: அக்னிவேசரின் சரக சம்ஹிதை 3ம் பாகம் பக்க எண்: 187


EGG WHITE

PREPARATION OF THE TRIAL DRUG

KARPOORA SILASATHU PARPAM (INTERNAL MEDICINE)

(Ref: Dr.K.Anbarasu, Agathiyar chendhooram - 300, moolamum uraiyum 1st edition, 1998, page no: 13 & 14)

கற்பூரச் சிலாசத்து பற்பம்

கேளப்பா கற்பூர சலாசத் துத்தான்
கெடியாக பலமொன்று பொரிகா ரந்தான்
நாளப்பா பலமொன்று ரெண்டுங் கூட்டி
நலமான பருத்தி நீறு துத்தி வேர்த்தோல்
நீளப்பா பாங்காகக் ககூடாயம் செய்து
நிசமாக விட்டாட்டு ஒரு நாள் மட்டும்
மாளப்பா அஞ்செருவிற் புடத்தை போடு
ஆறியெடு கல்வத்தி லாட்டு தானே

ஆட்டத்தூ ளாகுமடா வெண்கருவை விட்டு
அரைத்துவில்லை செய்துரவி யிலுலர்த்திக் கொண்டு
மாட்டவே அஞ்செருவிற் புடத்தைப் போடு
வாகாக அரைத்து அஞ்சு தரமு மானால்
நாட்டவே வெண்பொடியாம் பதனம் பண்ணு
நலமான பெருவயிறும் மகோதரங்கள்
ஊட்டவே நீர்ச்செரிப்பு கல்லடைப்பு
ஓடுமப்பா வெடியுப்பு சுன்ன நீரே

INGREDIENTS:

- Purified karpoora silasathu - 1palam(35gms)
- Purified Vengaram - 1palam(35gms)
- Ash of paruthi plant - Required amount
- Stem bark of Thutthi - Required amount
- Egg white - Required amount

METHOD OF PURIFICATION

- **Karpoora silasathu**: Purified by boiling it with tender coconut. Then it is washed in the water.
- **Vengaram:** Purified by fried in a mud vessel until the water content of it evaporates.

- **Thutthi ver thol:** Wash the root with running water and dry it.

METHOD OF PREPARATION:

Preparation of decoction:

A decoction is prepared by mixing ash of paruthi plant (*Gossypium herbaceum*) and the root bark of Thutthi (*Abutilon indicum*) in required amount of water.

Then, purified karpoora silasathu and Vengaram are taken and put into the Stone Martar and grinded by using the above said decoction for 24hrs. Then it is made into small Tablets (villai) and dried. Then it is subjected to pudam by using 5 cow dung cakes. The drug is again grinded by using egg white and made into small tablets (villai) and dried. Then it is again subjected into pudam by using 5 cow dung cakes. The above said procedure is repeated for 5 times.

Dosage: 130mg (twice/day) after food

Adjuvant: Radish juice

Indication: Renal calculus, Ascitis.

ASH OF PARUTHI, THUTHI

GRINDING WITH PARUTHI, THUTHI DECOCTION

PARUTHI DECOCTION + VILLAI

EGG

EGG WHITE

GRINDING WITH EGG WHITE

KARPOORA SILASATHU PARPAM

PROTOCOL

TITLE OF THE DISSERTATION PROJECT:

Pre clinical and clinical study on **AZHAL KALLADAIPPU** (Renal Calculi) and the drug of choice is **"KARPOORA SILASATHU PARPAM"** (Internal)

AIM:

To document the siddha drug **Karpoora Silasathu Parpam** in the treatment of **Azhal Kalladaippu** (Renal Calculi) by the standard process of evaluation of safety and efficacy of the drug.

OBJECTIVE:

1. Primary objective:

To evaluate the therapeutic efficacy of siddha drug **Karpoora**

Silasathu parpam (Internal) in the treatment of **Azhal Kalladaippu (Renal Calculi).**

2. Secondary objective:

1. To evaluate the safety profile (acute, long term toxicity studies) of this drug.

2. To study the effect of other co-factors such as age, sex and siddha parameters.

STUDY DESIGN & CONDUCT OF STUDY:

Study type: An open clinical trial.

Study place: OPD and IPD of Ayothidass Pandithar Hospital,
National Institute of siddha,
Tambaram sanatorium, Chennai-47.

Study period: 12 months.

Sample size: 40 patients.

TREATMENT:

Medicine name:

Karpoora silasathu parpam (Internal)

(Ref: Dr.K.Anbarasu, Agathiyar chendhooram - 300, moolamum uraiyum 1st edition, 1998, page no: 13 & 14)

Dosage: 130mg (twice/day) after food

Adjuvant: Radish juice

Route of Administration: Oral Route

Duration of the Drug Administration: 48 days

SUBJECT SELECTION:

As and when patients reporting at OPD of Ayothidoss Pandithar Hospital, NIS with symptoms of inclusion criteria will be subjected to screening test & documented using Screening Proforma.

SELECTION CRITERIA:

INCLUSION CRITERIA:

Patients who fulfilled any of the following criteria will be included in the study:

- Age: 20- 60Yrs
- Sex – Both male & female
- Patients who are having the classical symptoms of **abdominal pain & distensión, pain from loin to groin, pain in urethra, agonizing pain, dysuria, oliguria, yellow coloured urination, burning micturition, haematuria, nausea & vomiting.**
- Stone size**: ≥4mm &≤10mm**
- Patient with renal calculus detected on X-ray KUB or USG abdomen.
- History of Recurrence of Renal calculi.
- Patient willing to sign the informed consent stating that he/she will conscientiously stick to the treatment during 48days but can opt out of the trial of his/her own conscious discretion.
- Patients who are willing to take Ultrasonography Investigation (USG- abdomen / KUB) and provide blood for lab investigation.
- Crystals, Blood cells, Pus cells in urine which can be detected in the urine test.

EXCLUSION CRITERIA:

A potential subject who meets any of the following criteria will be excluded from participation in this study:

- Stone size >10mm
- Pregnancy and lactation
- Presence of any associated severe systemic illness, e.g.CA
- Patient taking any other lithotriptic agent
- History of Diabetes/ Hypertension
- History of Hepatic disease
- History of other Renal disease
- History of Cardiac disease
- History of drug/alcohol abuse
- History of taking treatment for any other ailments

WITHDRAWAL CRITERIA:

a. Intolerance to the drug & development of any serious adverse reactions during the trial period.
b. Poor patient compliance & defaulters.
c. Patient turned unwilling to continue in the course of clinical trial.
d. Increase in severity of symptoms.
e. Patient will not take medication regularly.

ASSESSMENTS AND INVESTIGATIONS:

a) clinical assessment
   Siddha assessment
b) Routine investigations:
   1. Modern parameters
   2. Siddha parameters

c) Specific investigations

a) CLINICAL ASSESSMENT:

- Abdominal pain
- Pain from Loin to groin
- Agonizing pain
- Pain in urethra
- Yellow coloured urination
- Burning Micturition
- Oliguria
- Dysuria
- Abdominal distension
- Nausea & vomitin
- Haematuria

SIDDHA ASSESSMENT:

1. Thinai (Land):

- Kurinchi (Hill areas)
- Mullai (Forest)
- Marutham (Fertile land)
- Neithal (Costal area)
- Paalai (Desert)

2. Paruvakaalam (Season)

- Kaar kaalam
- Koothir kaalam
- Munpani kaalam
- Pinpani kaala
- Elavenil kaalam
- Muthuvenil kaalam

3. Poripulankal:

- Mei (Skin)
- Vaai (Tongue)
- Kan (Eye)
- Mooku (Nose)
- Sevi (Ear)

4. Kanmedriyam and Gnanenthiriyam:

- Vaai (Buccal cavity)
- Kaal (Lower limbs)
- Kai (Upper limbs)
- Eruvaai (Anorectal region)
- Karuvaai (Uro-genital region)

5. Ezhu udal kaugal:

- Saram
- Senneer
- Uoon
- Kozhuppu
- Enbu
- Moolai
- Sukkilam /suronitham

6. Enn vagai thervu (Eight types of Examination):

- Naadi
- Sparisam
- Naa
- Niram
- Mozhi
- Vizhi
- Malam
- Moothiram

  -Neerkuri

  -Neikuri

b) ROUTINE INVESTIGATIONS:

❖ **Modern parameters:**

- Hb (gms/dl)
- Total RBC (million/cu.mm)
- Total WBC (cubic mm)
- Differential count: (%)
  - Polymorphs
  - Lymphocytes
  - Monocytes
  - Eosinophils
  - Basophils
- ESR(mm/hr)
- **Blood sugar level** Fasting (mg/dl)
  Post prandial (mg/dl)
  Random (mg/dl)
- **Lipid profile** Total cholesterol (mg/dl)
  HDL (mg/dl)
  LDL (mg/dl)
  VLDL (mg/dl)
  TGL (mg/dl)
- **Bleeding time -** /min
- **Clotting time -** /min
- **Renal function test** - Blood Urea (mg/dl)
  Serum total Creatinine (mg/dl)
  Uric acid (mg/dl)
- **Liver function test** - Serum total bilirubin (mg/dl)
  Direct bilirubin (mg/dl)
  Indirect bilirubin (mg/dl)
  SGOT (IU/L)
  SGPT (IU/L)
  Serum Alk.phosphotase (kingÅ units)

Serum calcium (mg/dl)

Serum phosphorus (mg/dl)

Total protein (mg/dl)

Serum albumin (mg/dl)

Serum globulin (mg/dl)

Serum fibrinogen (g/dl)

- **Urine**: Albumin
  Sugar (fasting& post prandial)
  Deposits
  Bile salts
  Bile pigments
  Urobilinogen
  Culture and sensitivity
- **Motion**: Ova
  Cyst
  Occult blood

❖ **Siddha parameters:**

- **Malam** - Niram:
  - Elakal / Erukal:
  - Muraigal (Times / day) kalappu:

- **Moothiram** (**urine**):
  - ✓ Neerkkuri (urine signs): neikkuri:
    i. Niram:
    ii. Edai:
    iii. Manam:
    iv. Nurai:
    v. Enjal

C) SPECIAL INVESTIGATIONS:

- Ultrasonography - Abdomen/ KUB
- X-ray- KUB

STUDY ENROLLMENT:

- In this clinical trial, patients reporting at NIS OPD with the clinical symptoms of abdominal pain, pain from loin to groin, agonizing pain, pain in urethra, dysuria, oliguria, yellow coloured urination, burning micturition, abdominal distension, haematuria, nausea & vomiting will be examined clinically for enrolling in the study based on the inclusion(including USG/KUB) and exclusion criteria.
- The patients enrolled in this study will be informed (Form IV) about the objective of
  the study, trial drug, possible outcomes in their own language and terms understandable to them.
- After ascertaining the patients willingness, informed consent will be obtained in the consent form (Form IV-A).
- All these patients will be given unique registration card in which patients Registration number of the study, Address, Phone number and Doctors phone number etc. so as to report easily and any adverse reaction arise.
- Complete clinical history, complaints and duration, examination findings-- all will be recorded in the prescribed Proforma in the history and clinical assessment forms separately. Screening Form- I will be filled up; Form I-A,

  Form –II and Form –III will be used for recording the patients' history, clinical

  examination of signs and symptoms and laboratory investigations respectively.
- Patients will be advised to take the trial drug and appropriate dietary advice (Form IV-D) would be given according to the patients perfect understanding.

CONDUCT OF THE STUDY:

The trial drug "**KARPOORA SILASATHU PARPAM**" is given continuously for 48 days. For OP patients, they should visit the hospital once in 7days. At each clinical visit clinical assessment is done and prognosis is noted. For IP patients the drug is provided daily and prognosis is noted.

Laboratory investigations & ultrasonography investigation are done 0^{th} day & 49^{th} day of the trial. For IP patients, who is not in a situation to stay in the hospital for a long time is advised to attend the OPD for further continuation of the treatment.

During the course of the treatment, patient is advised not to take tamarind, tea, coffee, non-veg, tomato, cabbage, cauliflower etc., and advised to take the diet as given in Form IV- D.

Follow-up:

After the end of the treatment, the patient is advised to visit the OPD for another 2months for follow-up.(medicines will not be given) If any of the trial patient who fails to collect the trial drug on the prescribed day but wants to continue in the trial, from the next day or two, he/ she will be allowed, but defaulters of one week and more will not be allowed to continue and be withdrawn from the study with fresh case being inducted.

DATA MANAGEMENT:

- After enrolling the patient in the study, a separate file for each patient will be opened and all forms will be filed in the file. Study No. and Patient No. will be entered on the top of file for easy identification. Whenever the study patient visits OPD during the study period, the respective patients file will be taken and necessary recordings will be made at the assessment form or other suitable forms.

- The screening forms will be filed separately.

- The Data recordings will be monitored for completion by HOD, SRO (statistics) and the adverse event will be monitored by the members of the pharmacovigilance department of NIS . All forms will be further scrutinized in presence of Investigator by Sr.Research Officer (Statistics) for logical errors and incompleteness of data to avoid any bias. No modification in the results is permitted for unbiased reports.

OUT COME OF TREATMENT:

Study Outcome is mainly assessed by,

1. Clearance/ reduction in the size of renal calculus in X-ray KUB and USG abdomen.

2. Complete reduction of clinical symptoms and improvement in the lab investigations.

ADVERSE EFFECT/SERIOUS EFFECT MANAGEMENT:

If the trial patient develops any adverse reaction (acute renal colic i.e. severe pain caused by the kidney stone and associated with nausea, vomiting and fever) he/she will be referred to the pharmacovigilance department of NIS. The members of this department will assess the adverse event and recorded in the prescribed adverse reaction form. For any AE the investigator will be given the proper management in NIS OPD with free of cost.

STATISTICAL ANALYSIS:

All collected data will be entered into the computer and manually cross-checked the correctness of the data entry. The clinical symptoms and size of the stone will be analyzed by comparing the two point of data (before and after treatment) paired test and chi-square test will be employed to study the efficacy of treatment. Further, the effect of age and sex will also be analyzed.

ETHICAL ISSUES:

1. Informed consent will be obtained from the patient explaining in the understandable language to the patient.

2. After the consent of the patient (through consent form) they will be enrolled in the study.

3. Treatment will be provided free of cost.

4. USG- Abdomen will be taken in the NABL certified laboratories and charges will be borne by the Investigator.

5. No other external or internal medicines will be used. There will be no infringement on the rights of patient.

6. To prevent any infection, while collecting blood sample from the patient, only Disposable syringes, disposable gloves, with proper sterilization of lab equipments will be used.

7. The data collected from the patient will be kept confidentially. The patient will be informed about the clinical trial, diagnosis, treatment and follow-up.

8. The patients who are excluded [as per the exclusion criteria] are given proper treatment, with full care at NIS.

9. All adverse events occurs during the trial period will be recorded by the Pharmacovigilance department team members. If the event will be mild, the patient will be treated in NIS OPD. If the event will be severe the patient will be referred to the nearby Govt. hospital and take care of the patient until he/she will get recovery. The treatment will be provided free of cost.

20.0 ASSESSMENT FORMS:

Form - I Screening and Selection Proforma

Form - IA History Proforma on enrollment

Form - II Clinical Assessment on enrollment

Form - IIA Clinical Assessment during and after the trial

Form - III Laboratory investigations on enrollment during and after the trial.

Form - IV Information sheet

Form - IV A Consent form

Form - IV B Withdrawal form

Form - IV C Drug Compliance form

Form - IV D Dietary Advice form

Form - IV E Adverse Reaction form

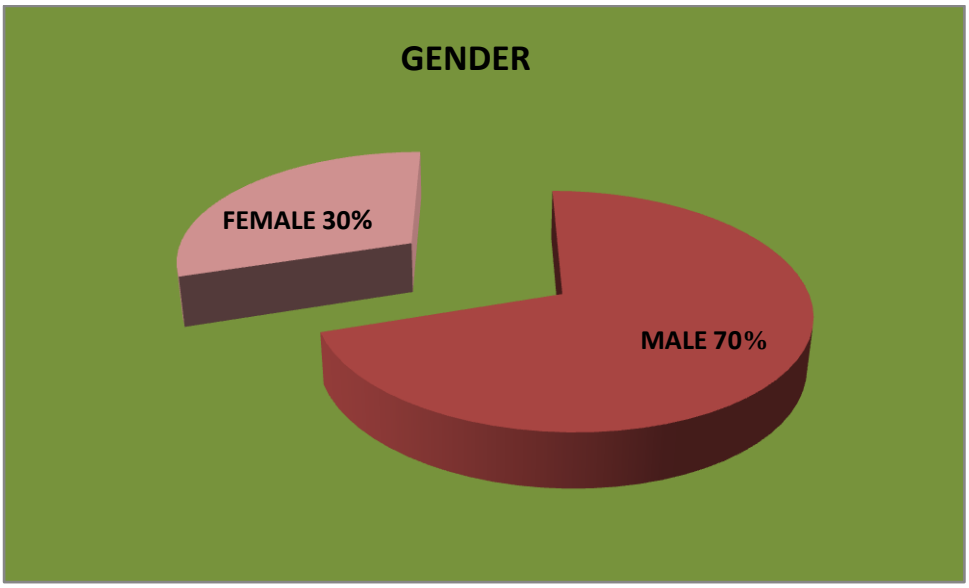
RESULTS AND OBSERVATIONS

Results were observed with respect to the following criteria:

1. Gender Distribution
2. Age Distribution
3. Gunam Distribution
4. Kaalam Distribution
5. Dietary Habits
6. Seasonal Variation
7. Thinai
8. Occupational Status
9. Treatment History
10. Chronicity of Illness
11. Clinical Features
12. Disturbances in Vatham
13. Disturbances in Pitham
14. Disturbances in Kabam
15. Kosangal
16. Naadi
17. Neerkuri
18. Neikkuri
19. Calculus in urinary system
20. Hydrouretronephrosis
21. Urine culture and sensitivity
22. Outcome - Result of USG abdomen
23. Before after treatment of clinical features
24. Result of Clinical features

DISTRIBUTION OF CASES BY GENDER

GENDER	NO. OF CASES	PERCENTAGE (%)
MALE	**28**	**70**
FEMALE	12	30
TOTAL	40	100

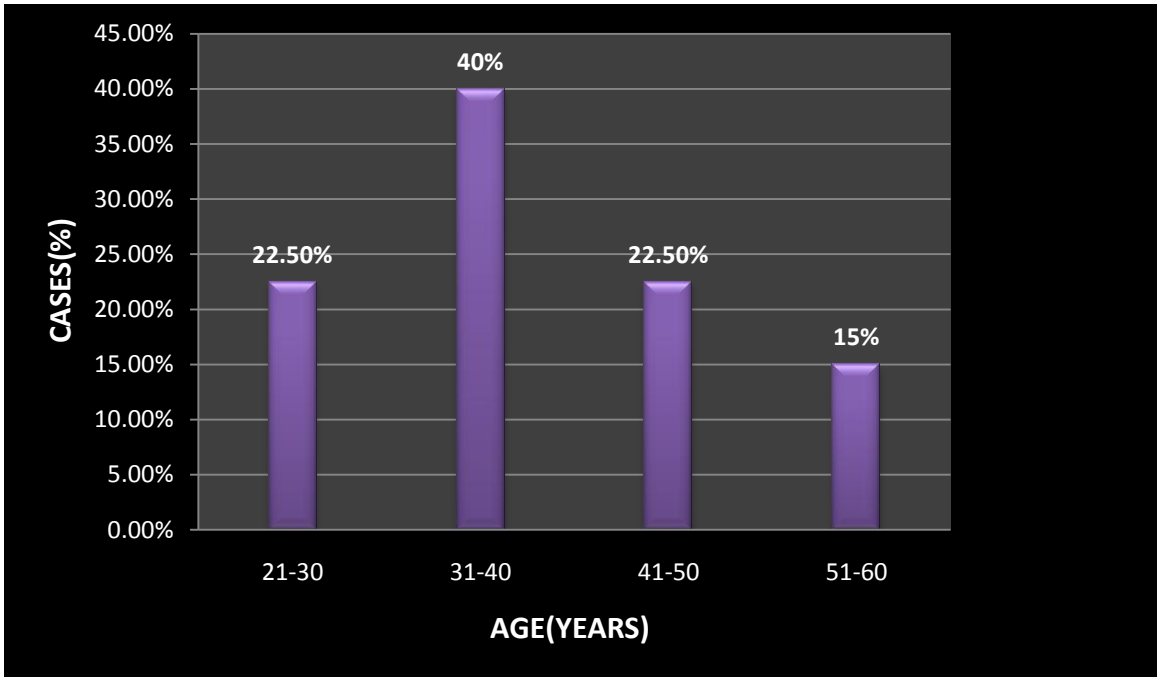


INFERENCE:

Among the 40 cases the prevalence of the disease was found to be higher in males. i.e. 70 % (28cases).

DISTRIBUTION OF CASES BY AGE

AGE (YEAR)	NO. OF CASES	PERCENTAGE %
21 to 30	9	22.5
31 to 40	**16**	**40**
41 to 50	9	22.5
51 to 60	6	15
TOTAL	40	100

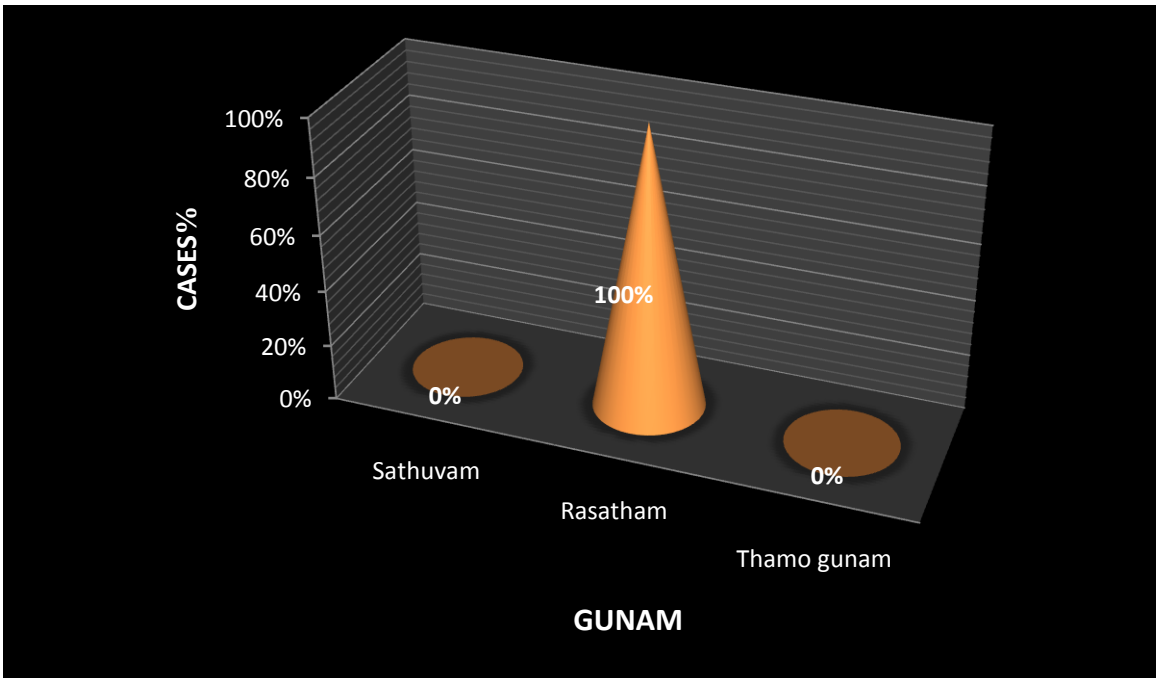


INFERENCE:

The prevalence of the disease was found to be higher in the age group 31 - 40 years. i.e, 16 cases (40%).

DISTRIBUTION OF CASES BY GUNAM

GUNAM	NO. OF CASES	PERCENTAGE (%)
Sathuvam	0	0
Rasatham	**40**	**100**
Thamo gunam	0	0

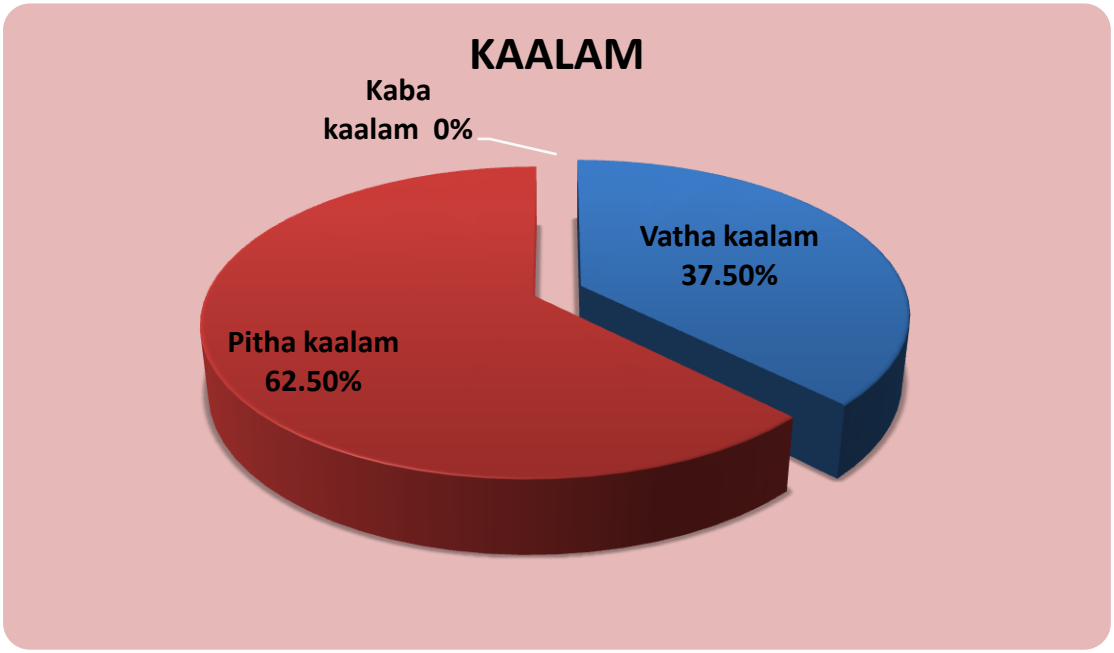


INFERENCE:

All the 40 cases (100 %) were found to posses Rasatha gunam.

DISTRIBUTION OF CASES BY KAALAM (LIFE SPAN)

KAALAM	NO. OF CASES	PERCENTAGE %
Vatha kaalam (upto 33years)	15	37.5%
Pitha kaalam (33 – 66 years)	**25**	**62.5%**
Kaba kaalam (66 – 100 years)	0	0%

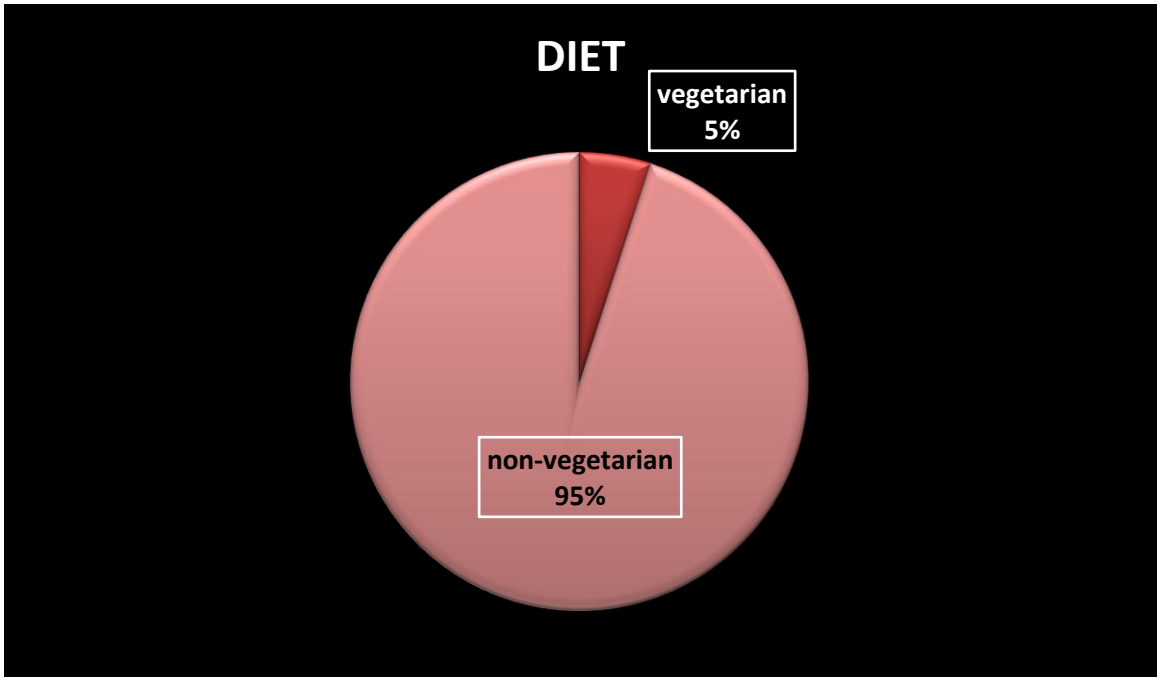


INFERENCE:

Out of 40 cases, 25 cases (62.5%) were found to be in Pitha kaalam i.e. between 33 - 66 years and 15 cases (37.5%) were in vatha kaalam.

DISTRIBUTION OF CASES BY FOOD HABITS

FOOD HABITS	NO. OF CASES	PERCENTAGE %
Vegetarian	2	5
Non- vegetarian	**38**	**95**
Total	40	100

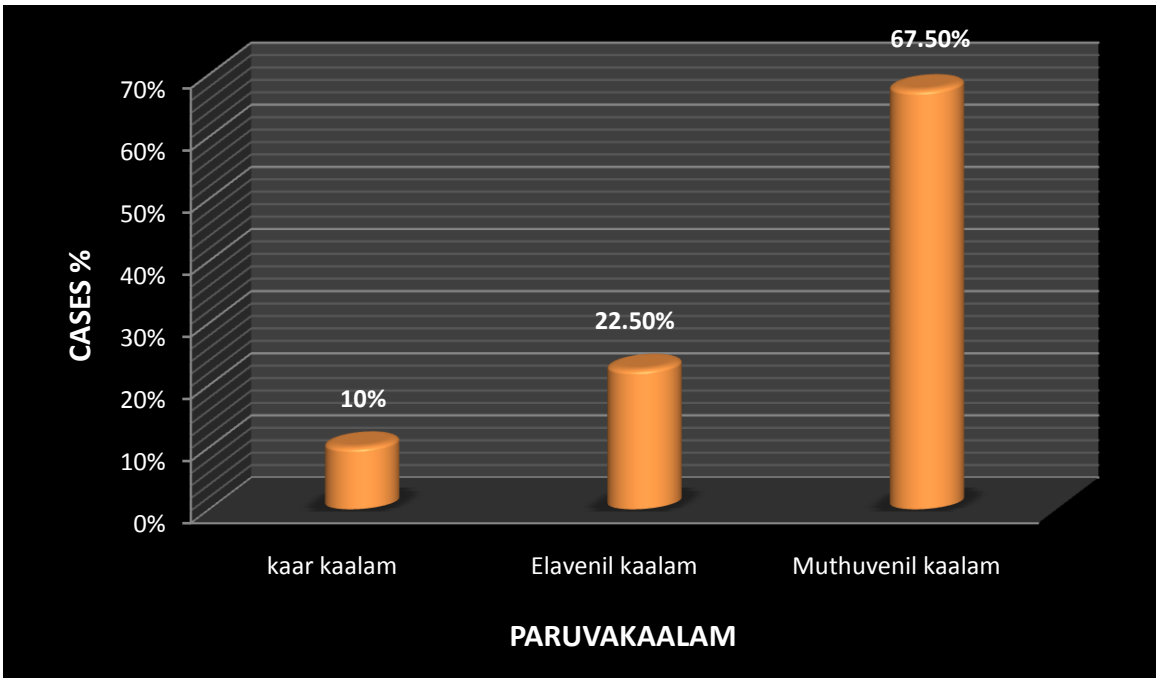


INFERENCE:

Among the 40 cases 38 cases (95%) were Non vegetarians and 2 cases (5%) were vegetarians.

DISTRIBUTION OF CASES BY PARUVAKAALAM (SEASON)

PARUVA KAALAM	NO. OF CASES	PERCENTAGE %
Kaar kaalam(Aug 17 - Oct 17)	4	10
Koothir kaalam(Oct 18 - Dec 15)	0	0
Munpani kaalam(Dec 16 - Feb 12)	0	0
Pinpani kaalam(Feb 13 - Apr 13)	0	0
Elavenil kaalam(Apr 14 - Jun 16)	9	22.5
Muthuvenil kaalam(Jun 17 - Aug 16)	**27**	**67.5**
Total	40	100

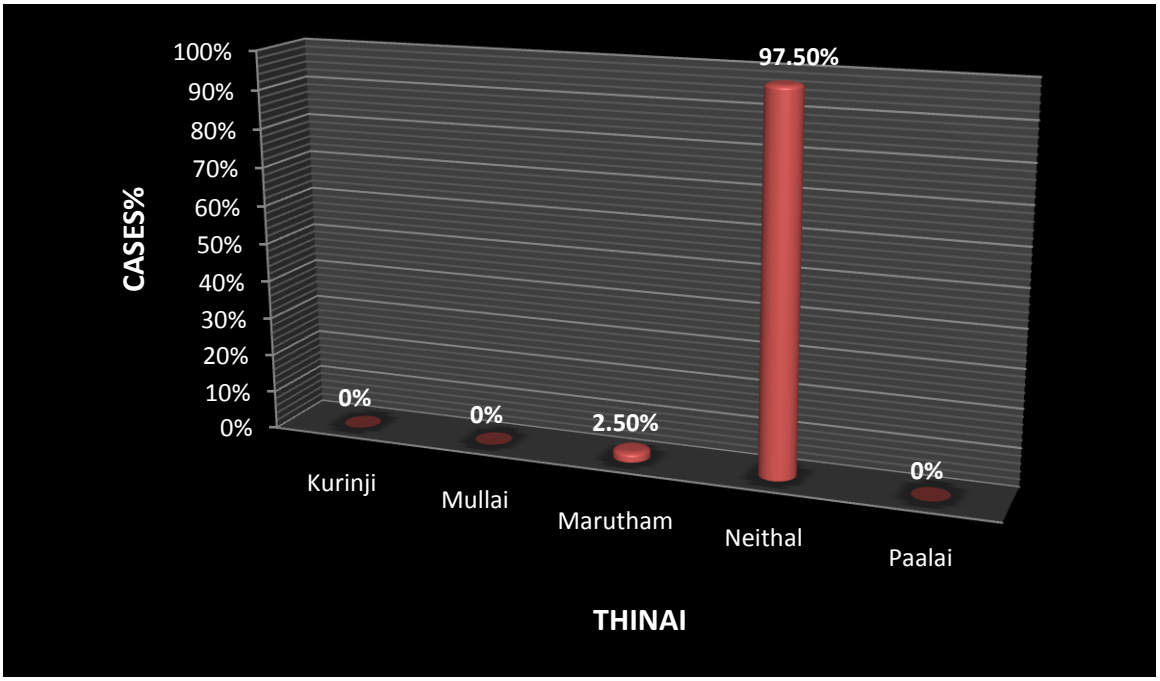


INFERENCE:

Among the 40 cases 27 cases (67.5 %) were admitted in Muthuvenil kaalam, 9 cases (22.5 %) were admitted in Elavenil kaalam and 4cases (10%) were admitted in kaar kaalam.

DISTRIBUTION OF CASES AS PER THINAI (LAND)

THINAI (LAND)	NO. OF CASES	PERCENTAGE %
Kurinji (Hill terrain)	0	0
Mullai (Forest range)	0	0
Marutham (Plain)	1	2.5
Neithal (Costal belt)	**39**	**97.5**
Paalai (Arid regions)	0	0
Total	40	100

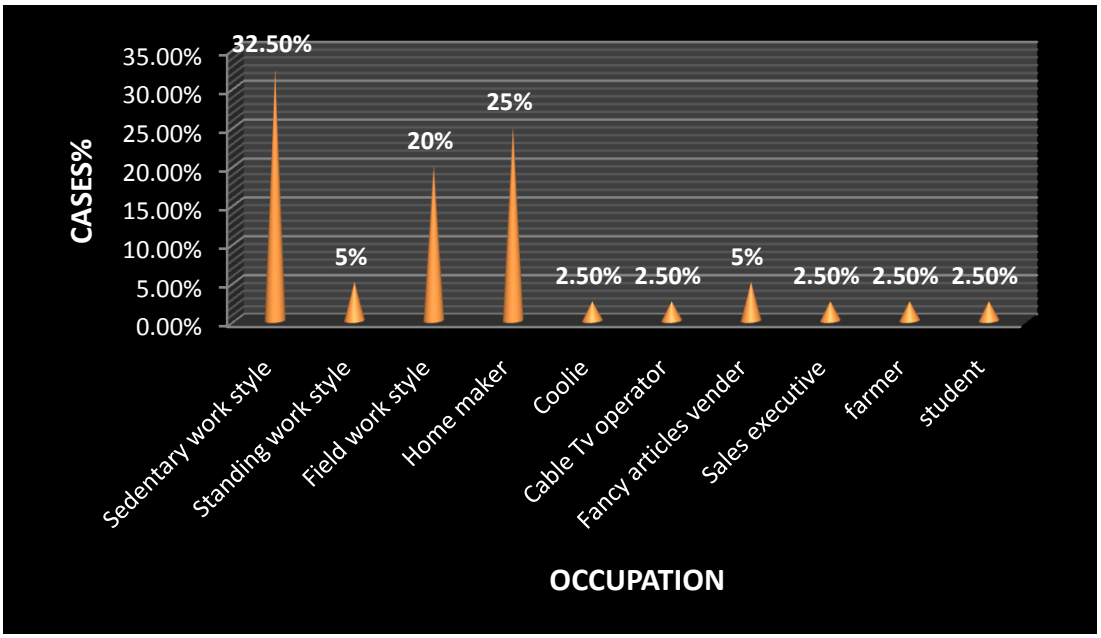


INFERENCE:

Among the 40 cases 39 cases (97.5%) were from Neithal thinai and 1 case (2.5%) was from Marutham thinai.

DISTRIBUTION OF CASES BY OCCUPATIONAL STATUS

S. NO	OCCUPATION	NO. OF CASES	PERCENTAGE %
1.	Sedentary work style	**13**	**32.5**
2.	Standing work style	2	5
3.	Field work style	8	20
4.	Home maker	10	25
5.	Coolie	1	2.5
6	Cable Tv operator	1	2.5
7.	Fancy articles vender	2	5
8.	Sales executive	1	2.5
9.	farmer	1	2.5
10.	student	1	2.5
Total		40	100

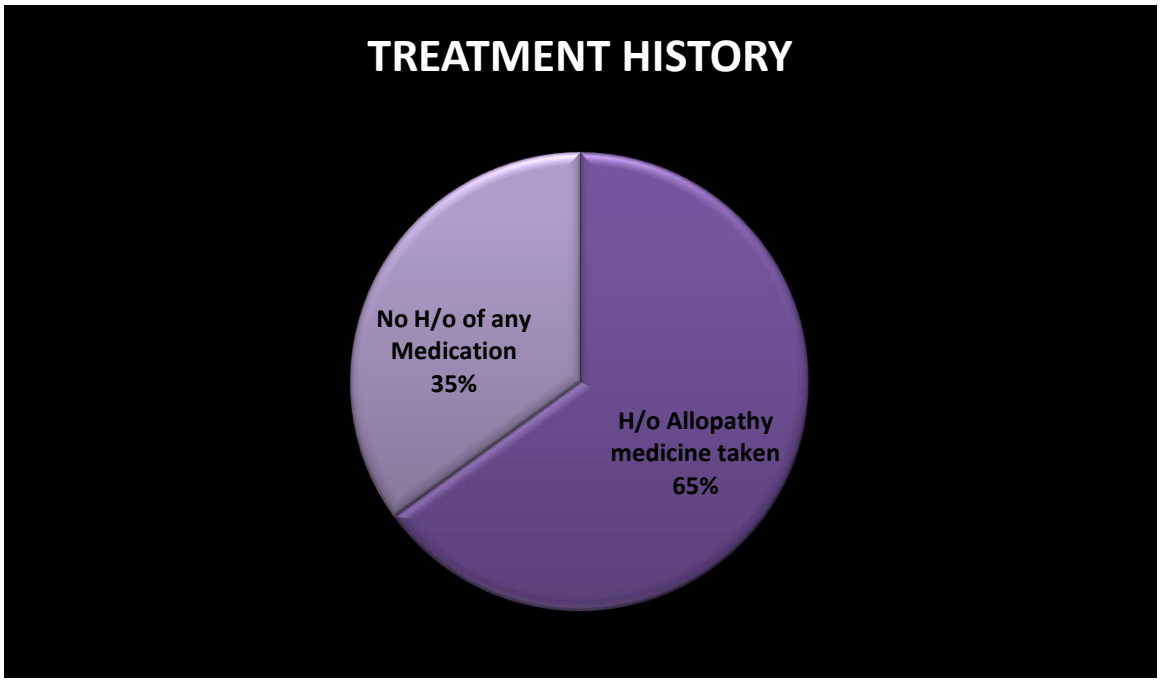


INFERENCE:

Among the 40 patients, 13cases (32.5%) were sedentary work style. 10cases (25%) were home maker. 8cases (20%) were field work and 2cases (5%) were standing work and fancy article vender. Rest of 1case (2.5%) were Coolie, Cable Tv operator, Sales executive and farmer.

TREATMENT HISTORY (BEFORE ADMITTED TO THE TRIAL)

TREATMENT HISTORY	NO. OF CASES	PERCENTAGE (%)
H/O Allopathy medicine taken	26	65
No H/O any Medication	14	35
Total	40	100

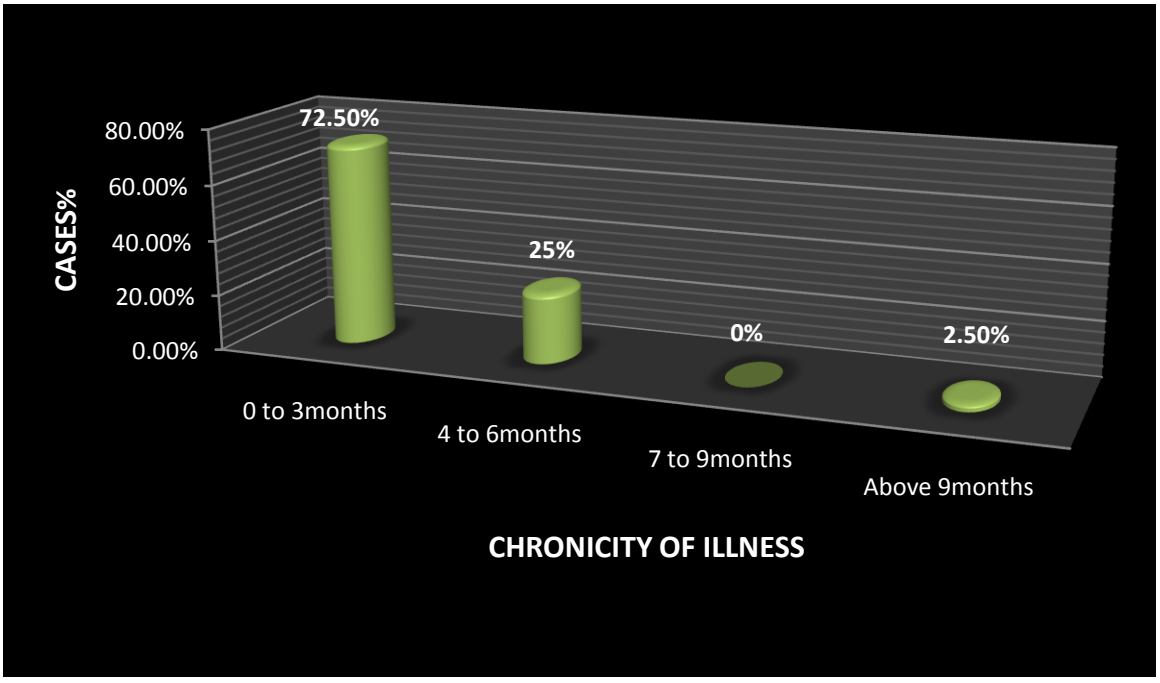


INFERENCE:

Among the 40cases, 26cases (65%) had taken allopathic treatment in the past and had discontinued the same. The rest of the 14cases (35%) had not taken any other drugs prior to enrolling for the study.

DISTRIBUTION OF CASES BY CHRONICITY OF ILLNESS

DURATION IN MONTHS	NO. OF CASES	PERCENTAGE %
0 to 3	**29**	**72.5**
4 to 6	10	25
7 to 9	0	0
Above 9	1	2.5
Total	40	100

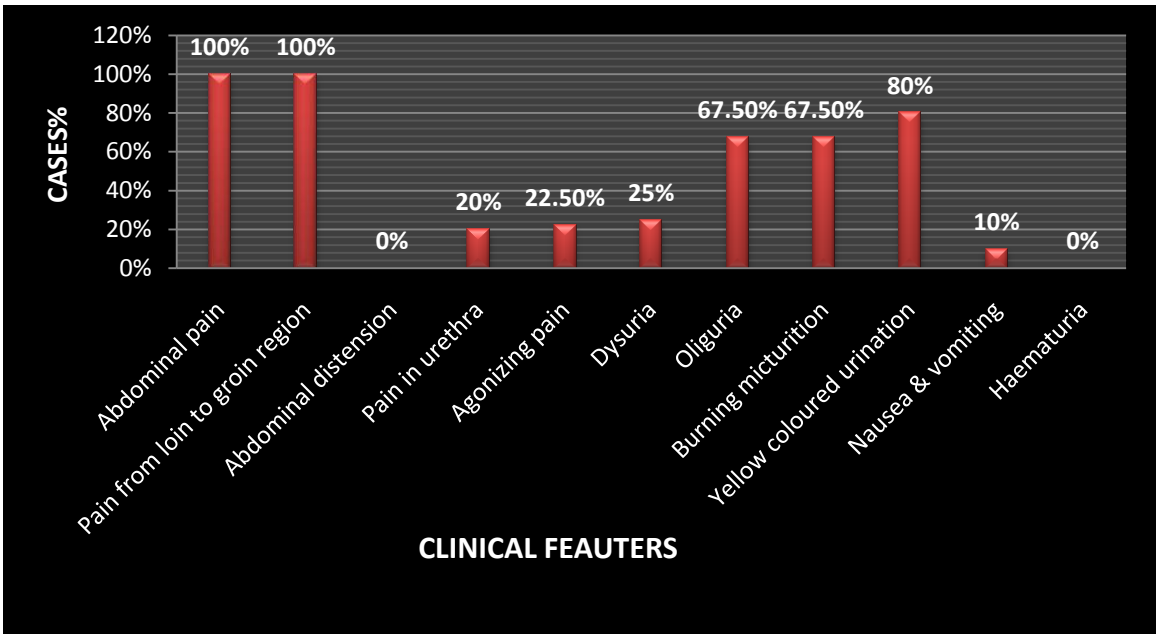


INFERENCE:

Among the 40 cases majority of them were 0 to 3 months in their duration of illness, i.e, 29 cases (72.5%), 10cases (25%) had the illness in 4 to 6 months and 1case (2.5%) was above 9 months.

DISTRIBUTION OF CASES AS PER CLINICAL FEATURES

CLINICAL FEATURES	NO. OF CASES	PERCENTAGE %
Abdominal pain	**40**	**100**
Pain from loin to groin region	**40**	**100**
Abdominal distension	0	0
Pain in urethra	8	20
Agonizing pain	9	22.5
Dysuria	10	25
Oliguria	27	67.5
Burning micturition	27	67.5
Yellow colored urination	32	80
Nausea & Vomiting	4	10
Haematuria	0	0

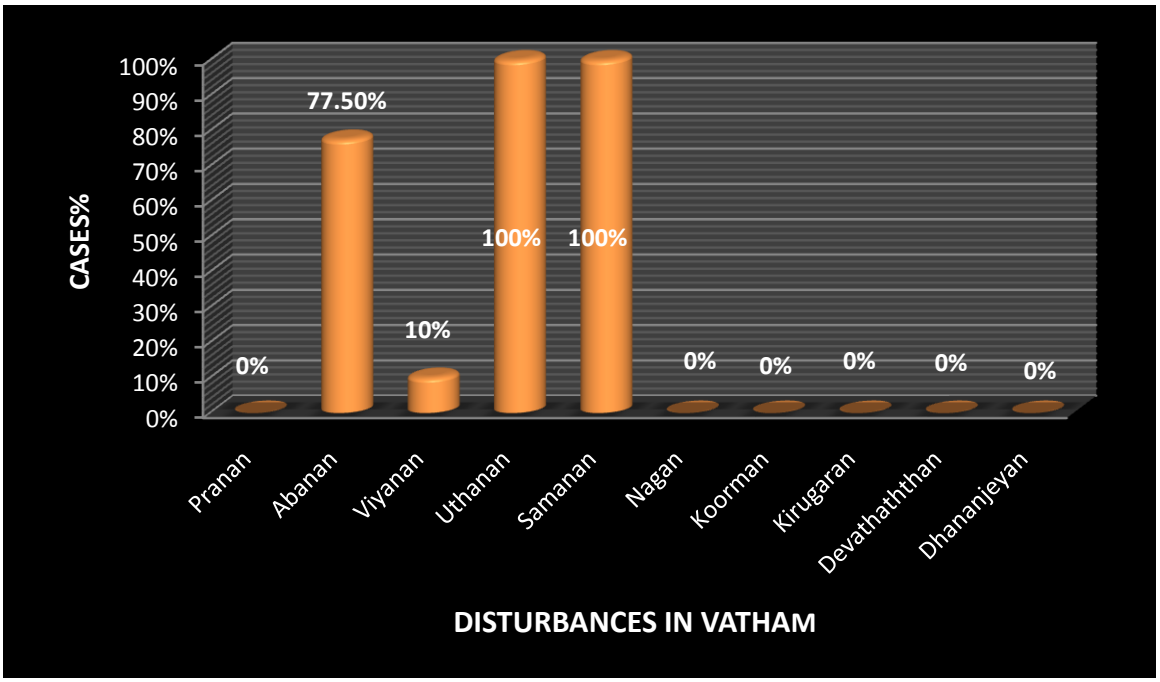


INFERENCE:

In clinical features, all the 40 cases (100%) had abdominal pain and pain from loin to groin region. 32cases(80%) had yellow coloured urination. 27cases (67.5%) had oliguria and burning micturition. 10cases (25%) were affected by dysuria. 9cases (22.5%) were affected by agonizing pain. 8cases (20%) had pain in urethra and 4cases (10%) had nausea and vomiting.

DISTURBANCES IN VATHAM

VATHAM	NO. OF CASES	PERCENTAGE (%)
Pranan	0	0
Abanan	31	77.5
Uthanan	4	10
Viyanan	**40**	**100**
Samanan	**40**	**100**
Nagan	0	0
Koorman	0	0
Kirugaran	0	0
Devethathan	0	0
Dhananjeyan	0	0

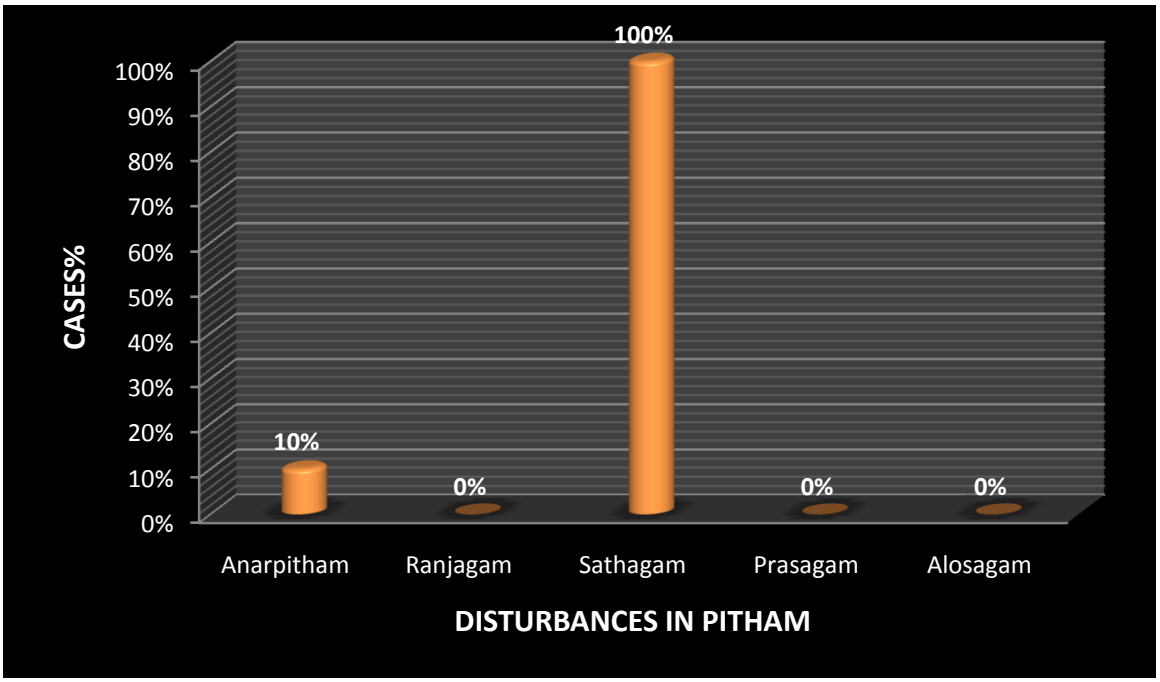


INFERENCE:

Out of 40 cases observed, viyanan was affected which resulted in abdominal pain and loin pain in all the 40 cases (100%) also samanan was affected in all the 40 cases (100%) because it controls the other types of vathas. Abanan was affected which resulted in oliguria in 31cases (77.5%)

DISTURBANCES IN PITHAM

PITHAM	NO. OF CASES	PERCENTAGE (%)
Anarpitham	4	10
Ranjagam	0	0
Sathagam	**40**	**100**
prasagam	0	0
Alosagam	0	0

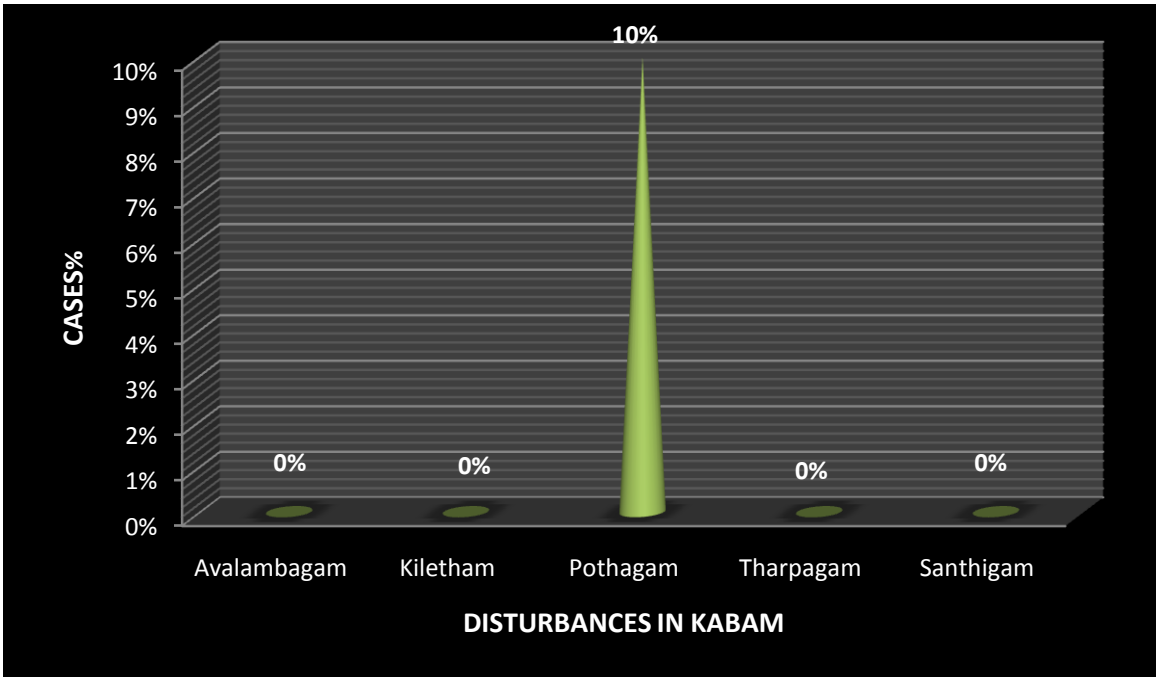


INFERENECS:

Out of 40 cases, saathaga pitham was affected in all the cases (100%) as a result the patients were unable to perform their routine duties. Anarpitham was affected in 4 cases (10%) which resulted in nausea and vomiting.

DISTURBANCES IN KABAM

KABAM	NO. OF CASES	PERCENTAGE (%)
Avalambagam	0	0
kilethagam	0	0
Pothagam	**4**	**10**
Tharpagam	0	0
santhigam	0	0

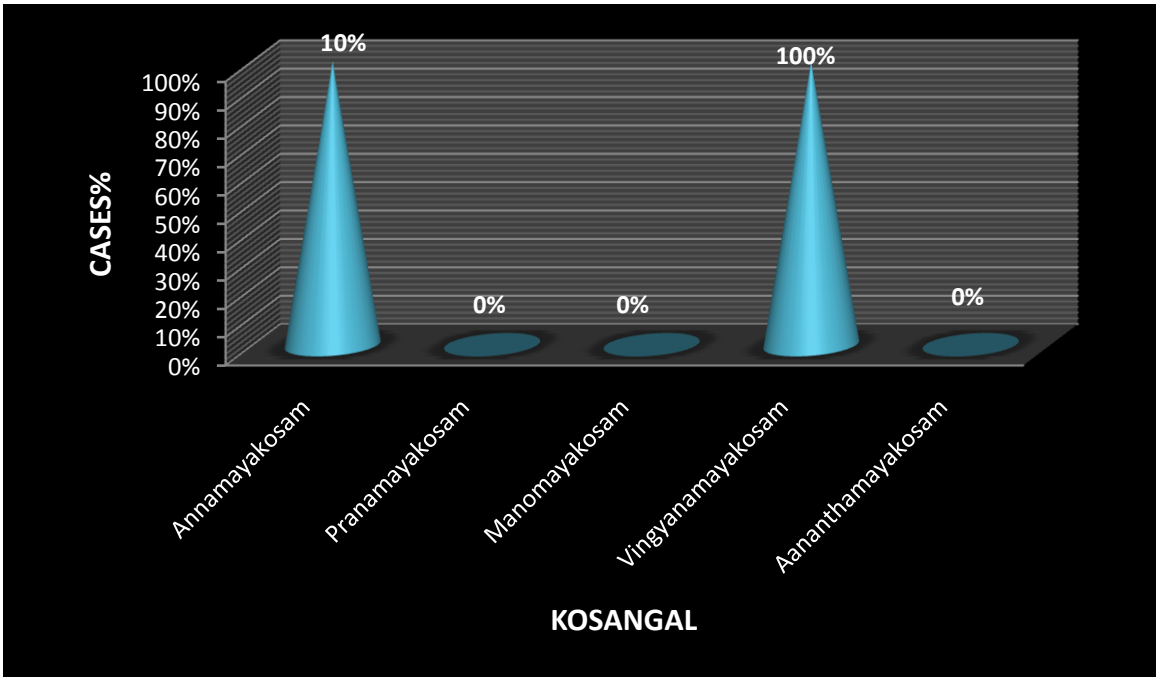


INFERENECS:

Out of 40 cases pothagam was affected in 4cases (10%) which resulted in nausea and vomiting.

KOSANGAL

KOSAM	NO. OF CASES	PERCENTAGE (%)
Annamaya kosam	4	10
Pranamaya koosam	0	0
Manomaya kosam	0	0
Vignanamaya kosam	**40**	**100**
Aananthamaya kosam	0	0

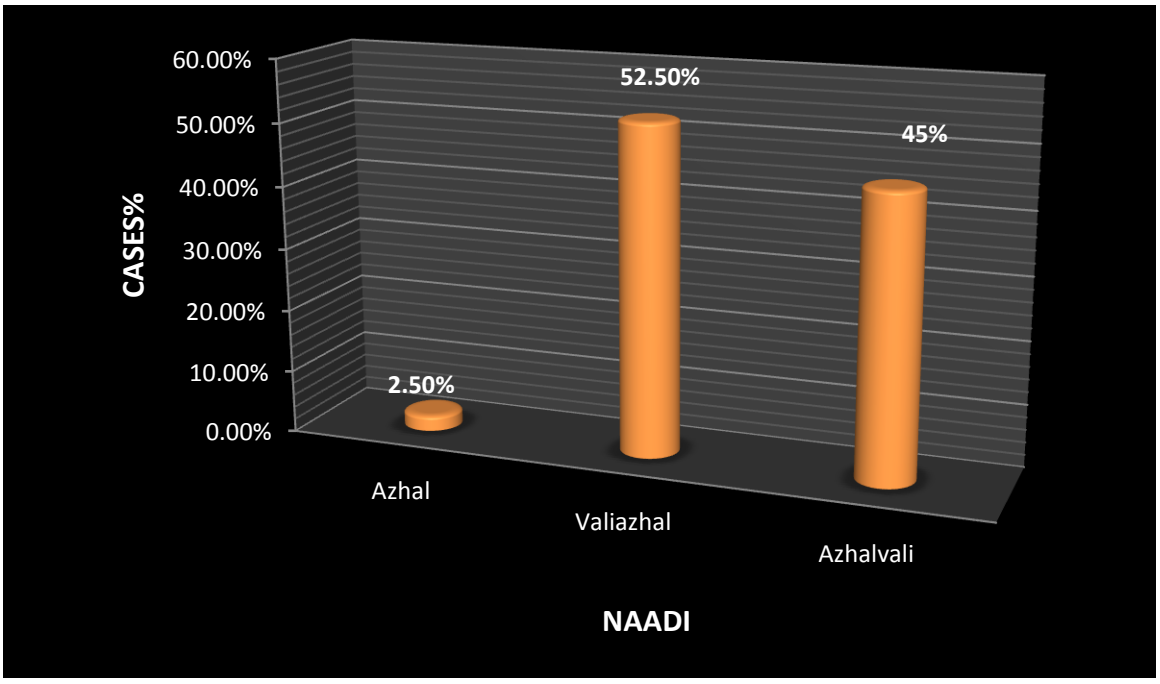


INFERENECS:

Out of 40 cases, vingyanamayakosam was affected in all the 40 cases (100%) which resulted in abdominal pain and loin pain and 4cases (10%) was affected in Annamaya kosam which resulted in nausea and vomiting.

DISTRIBUTION OF CASES BY NAADI

NAADI	NO. OF CASES	PERCENTAGE %
azhal	1	2.5
Valiazhal	**21**	**52.5**
Azhalvali	18	45
Total	40	100

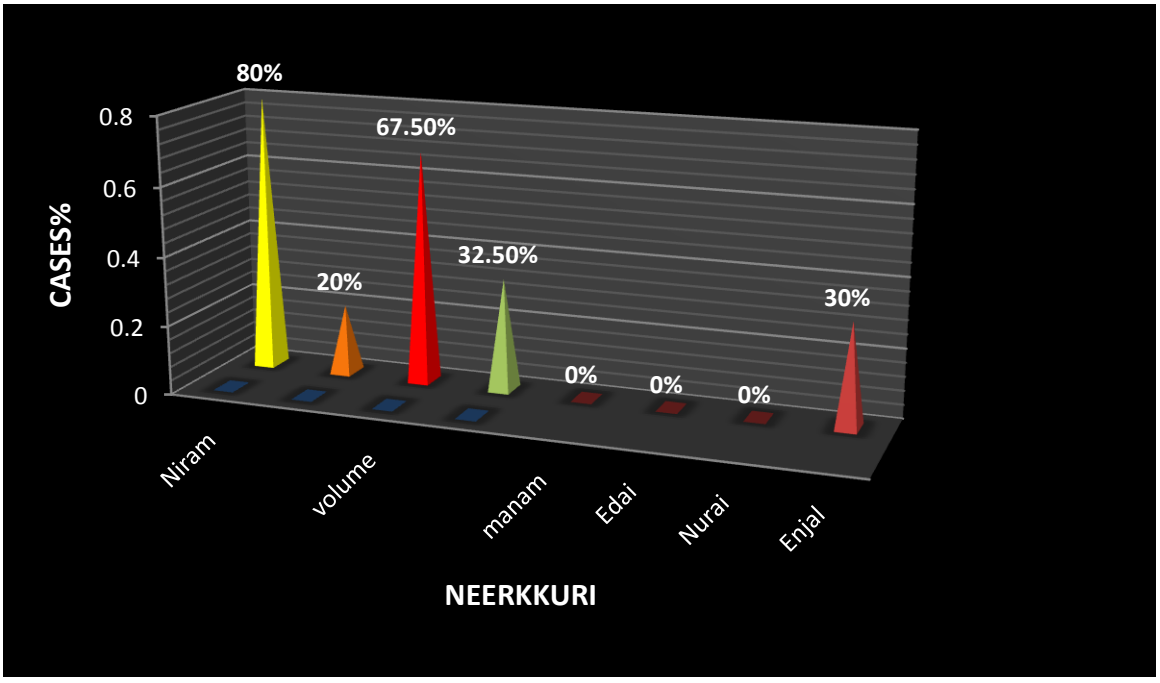


INFERENCE:

Among the 40 cases vali azhal naadi was felt in 21 cases (52.5 %), azhal vali naadi was felt in 18 cases (45 %); azhal naadi was felt in 1 case (2.5 %).

DISTRIBUTION OF CASES BY NEERKURI (URINE ANALYSIS)

NEERKURI		NO. OF CASES	PERCENTAGE %
Niram	Yellow colour	32	80
	Straw colour	8	20
volume	reduced	27	67.5
	normal	13	32.5
Manam		0	0
Edai		0	0
Nurai		0	0
Enjal (Deposites)		12	30

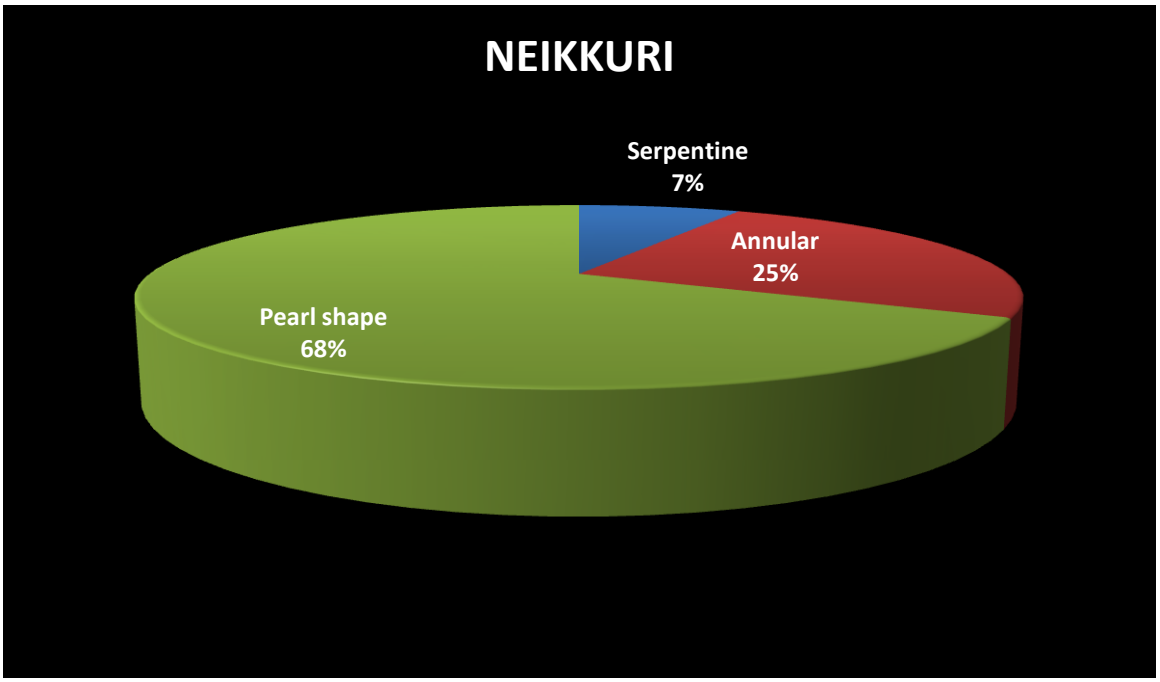


INFERENCE:

Out of 40 cases, yellow coloured was observed in 32 cases (80%) and straw coloured was in 8cases (20%). The volume of urine was reduced amount in 27 cases (67.5%); rest of 13 cases (32.5%) had normal urine volume. Enjal was found to be in 12 cases (30%) in the presence of bacterial infections. No other changes were observed in Manam, Edai and Nurai.

DISTRIBUTION OF CASES BY NEIKKURI (OIL SIGN IN URINE)

NEIKKURI	NO. OF CASES	PERCENTAGE %
Serpentine	3	7.5
Annular/ring	10	25
Pearl shape	27	67.5
Total	40	100

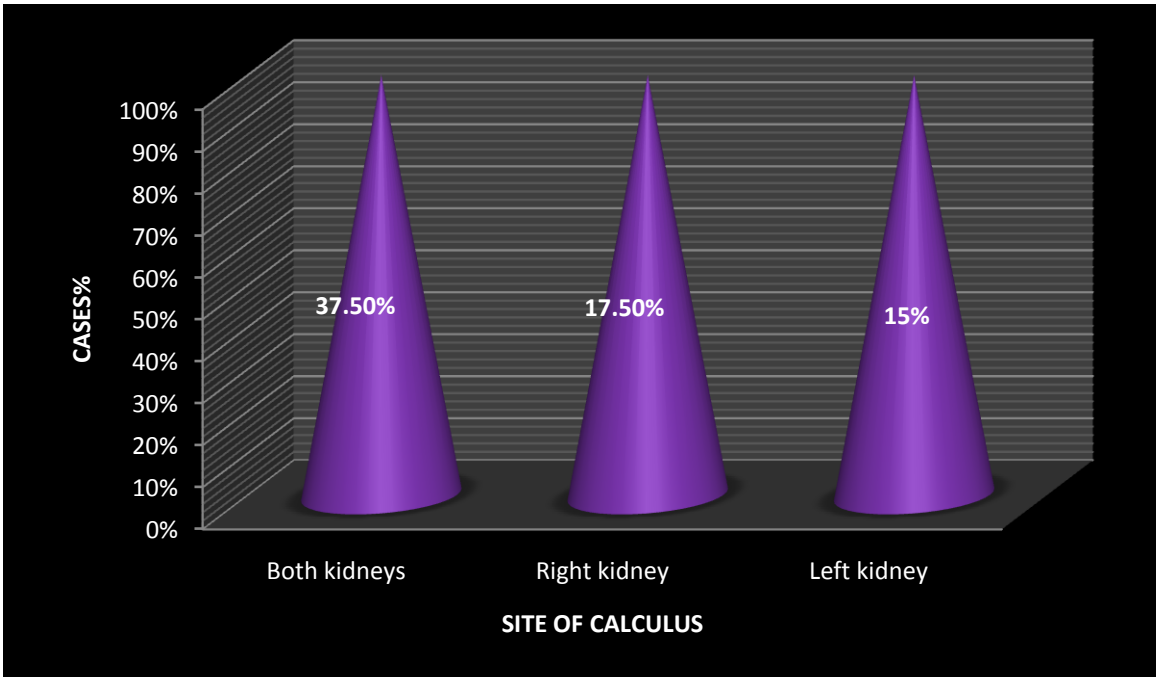


INFERENCE:

Among the 40 cases, in 27cases (67.5%) the neikkuri was observed as pearl like. In 10cases (25 %) the neikkuri was observed as Annular like and in 3cases (7.5%) neikkuri was observed as serpentine like.

DISTRIBUTION OF CALCULUS IN URINARY SYSTEM

SITE OF CALCULUS	NO. OF CASES	PERCENTAGE %
Both kidneys	15	37.5
Right kidney	7	17.5
Left kidney	6	15
Ureter	12	30

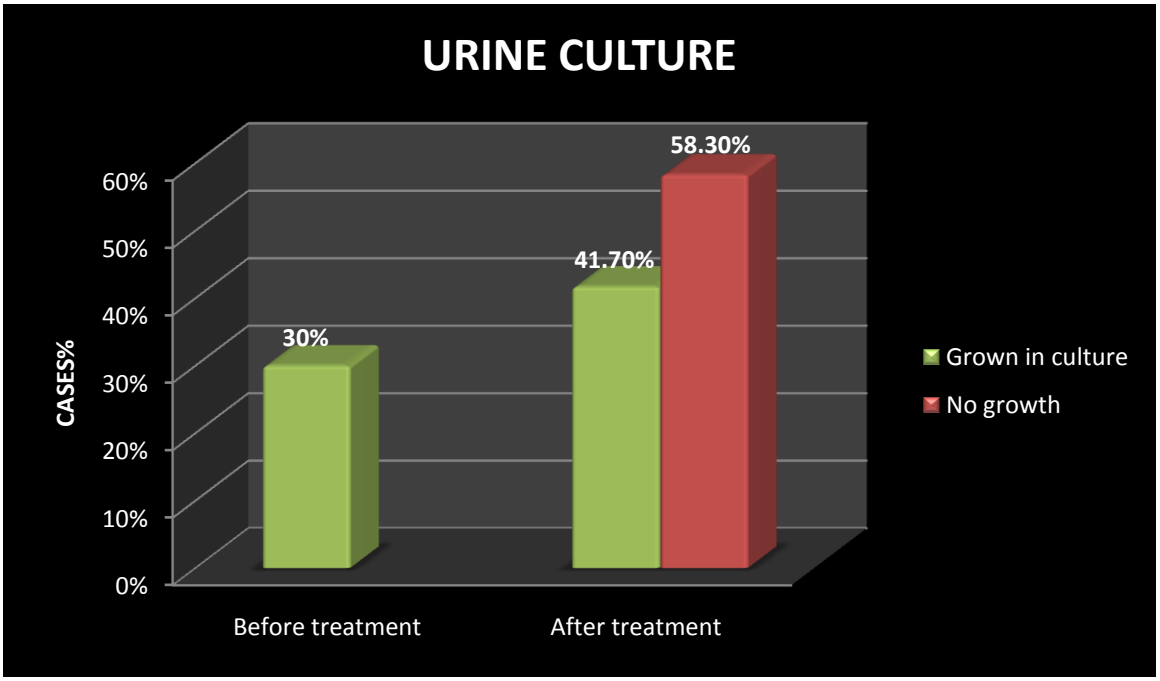


INFERENCE:

Out of 40 cases, 15 cases (37.5%) had bilateral renal calculi, 13 cases (32.5%) had unilateral renal calculi, out of them 7cases (17.5%) in right kidney and 6 cases (15%) in left kidney. 12cases (30%) had ureteric calculi.

URINE CULTURE AND SENSITIVITY

CULTUREAND SENSITIVITY		NO. OF CASES	PERCENTAGE (%)
Before treatment		12	30
After treatment	No growth	**5/12**	**41.7**
	Grown in culture	7/12	58.3

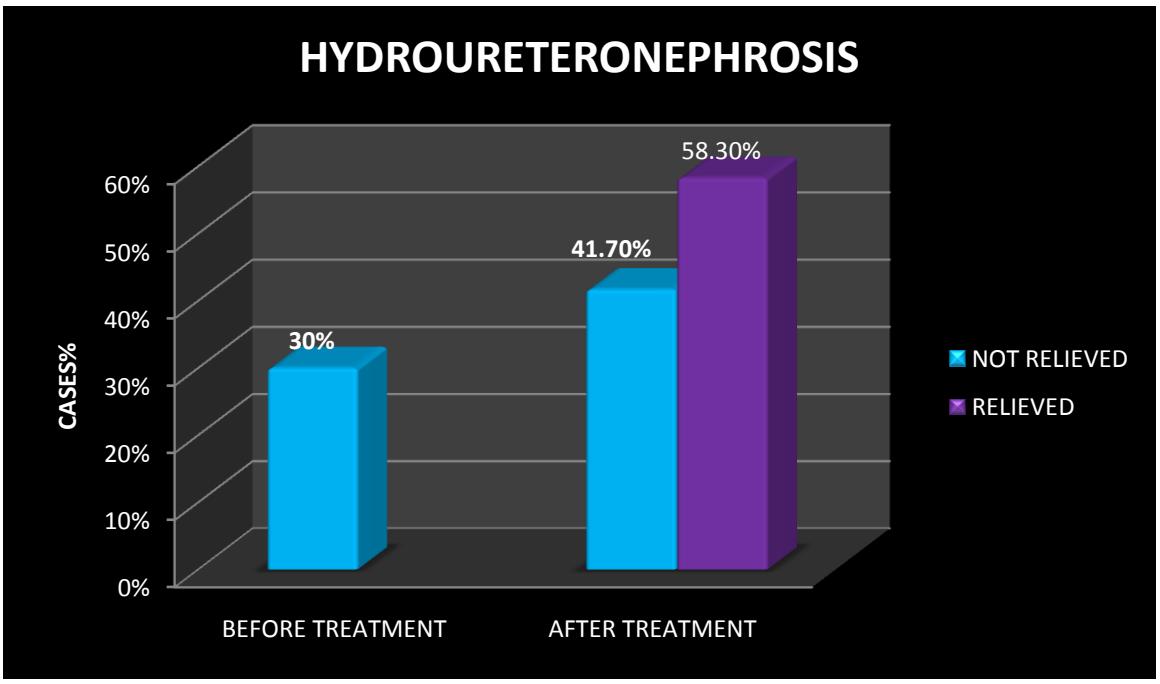


INFERENCE:

Among the 40 cases, 12cases (30%) had Bacterial infections in urine culture before treated with trial drug. Out of them 5cases (41.7%) were found to be no bacterial infections in urine culture after the completion of the trial drug treatmen and 7 cases (58.3%) had grown in culture.

HYDROURETERONEPHROSIS

HYDROURETERONEPHROSIS		NO. OF CASES	PERCENTAGE (%)
Before treatment		12	30
After treatment	**Relieved**	**7/12**	**58.3**
	Not relieved	5/12	41.7



INFERENCE:

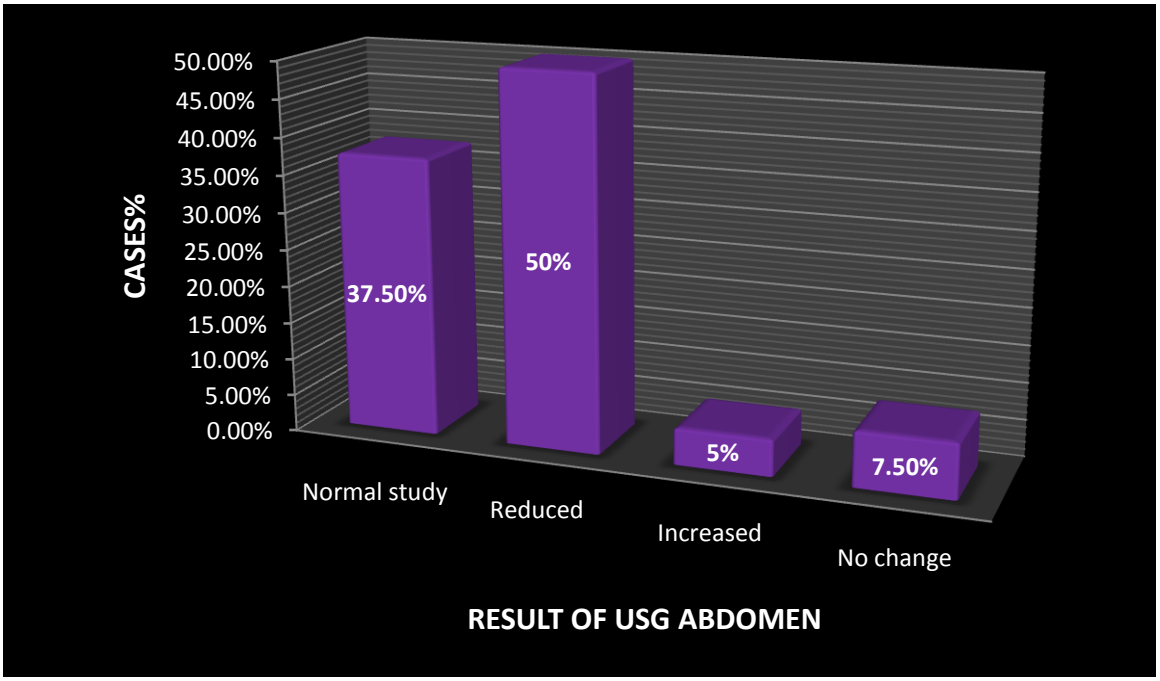
Before treated with trial drug 12 cases (30%) had hydroureteronephrosis. Among them, 7cases (58.3%) were relieved from hydroureteronephrosis after the treatment with trial drug and 5 cases (41.7%) were not relieved.

OUTCOME MEASUREMENTS

1. PRIMARY OUTCOME OBSERVATION

RESULT OF USG ABDOMEN AFTER TREATMENT

RESULT	NO. OF CASES	PERCENTAGE %
Normal study	**15**	**37.5**
Reduced	**20**	**50**
Increased	2	5
No change	3	7.5
Total	40	100



INFERENCE:

Out of 40 cases, 15 cases (37.5%) showed normal study, i.e. Stone completely dissolved. 20cases (50%) showed reduce its size and number. 2cases (5%) showed increase in size and 3cases (7.5%) showed no changes in size.

IMPROVEMENT OF USG ABDOMEN

RESULT	NO. OF CASES	PERCENTAGE %
Good	**35**	**87.5**
Poor	5	12.5
Total	40	100

Based on the above result of **USG abdomen** after treatment,

87.5% showed Good results and **12.5%** showed poor results.

Good - Normal study (stone completely dissolved)

Reduced its number (more than 2 / multiple)

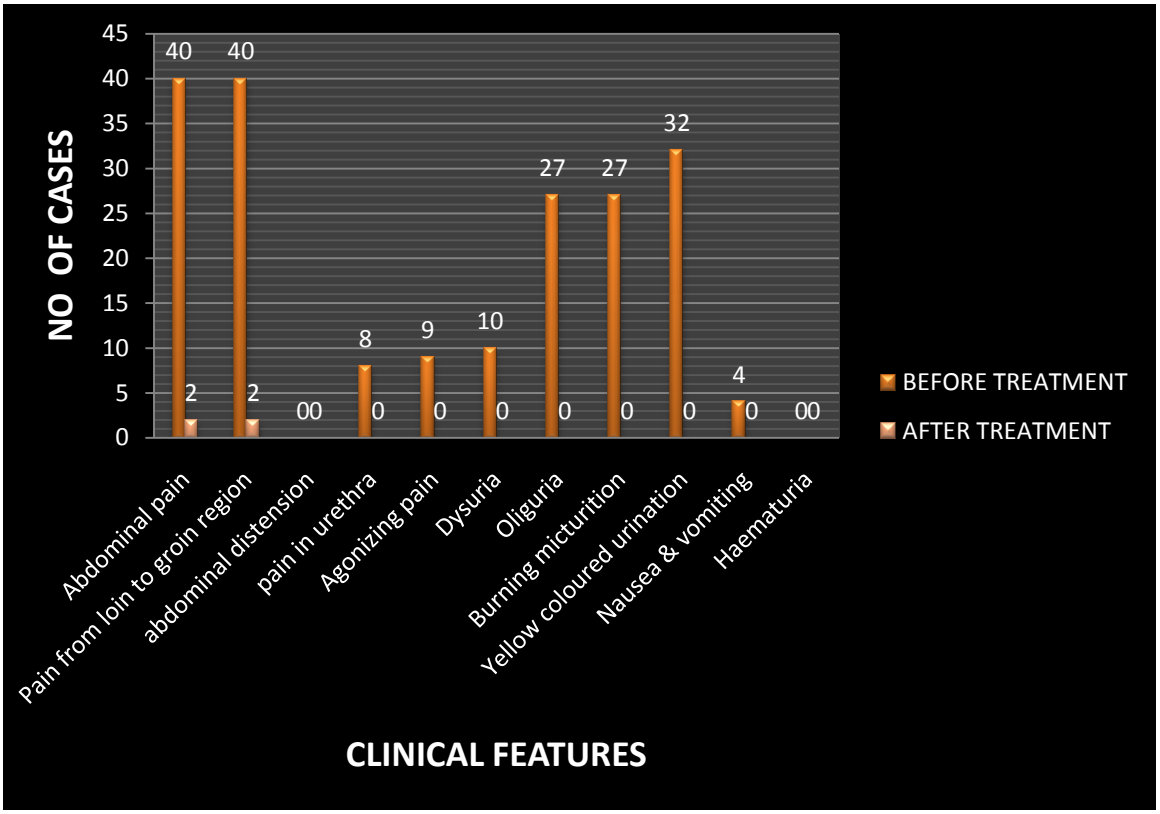
Reduced its size.

Poor - No change in size and increase in size

2. SECONDARY OUTCOME OBSERVATION

CLINICAL FEATURES BEFORE AND AFTER TREATMENT

CLINICAL FEATURES	BEFORE TREATMENT	AFETR TREATMENT
Abdominal pain	40	2
Pain from loin to groin region	40	2
Abdominal distension	0	0
Pain in urethra	8	0
Agonizing pain	9	0
Dysuria	10	0
Oliguria	27	0
Burning micturition	27	0
Yellow colored urination	32	0
Nausea and Vomiting	4	0
Haematuria	0	0

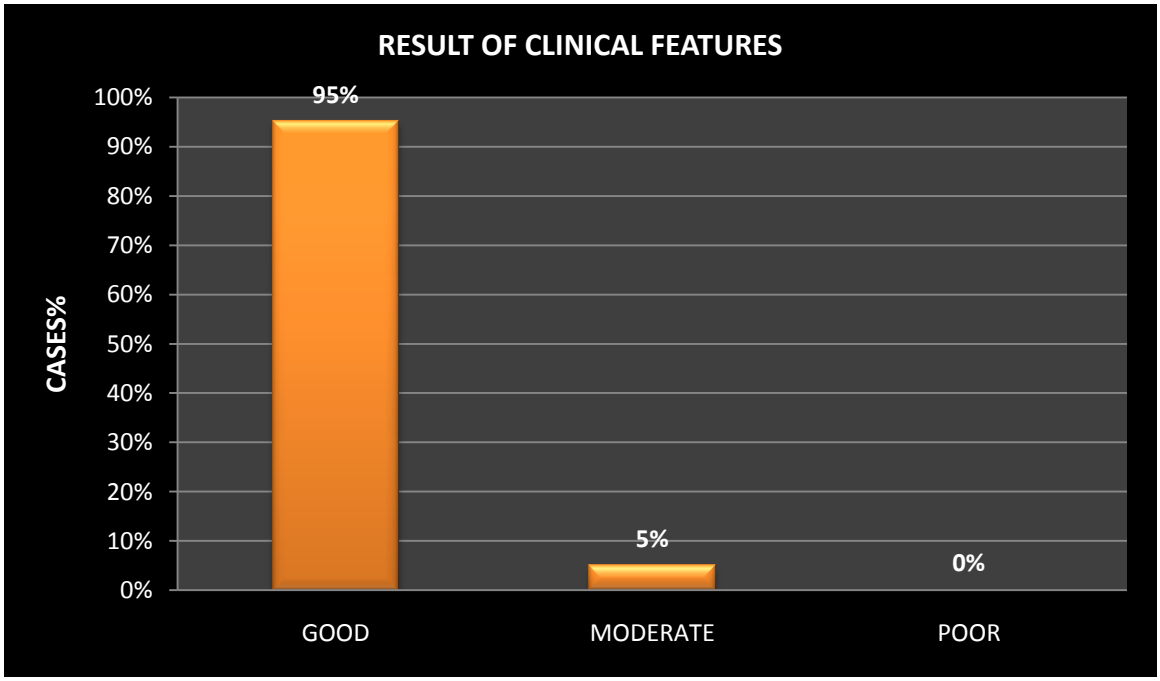


INFERENCE:

After treatment 38 cases (95%) had relieved from abdominal pain and loin pain. Rest of the other symptoms such as abdominal distension, pain in urethra, agonizing pain, dysuria, oliguria, yellow coloured urination, burning micturition, nausea and vomiting, haematuria were relieved in 100% of cases.

RESULT OF CLINICAL FEATURES AFTER TREATMENT

RESULT	NO. OF CASES	PERCENTAGE %
Good	**38**	**95**
Moderate	2	5
Poor	0	0
Total	40	100



Good - Symptoms completely relieved.

Moderate - Symptoms reduced.

Poor - Symptoms not reduced.

INFERENCE:

Out of the 40 cases good improvement was observed in 38 patients (95%), moderate improvement in 2 patients (5 %).

STATISTICAL ANALYSIS

Mean and Standard deviation was used to test the significance of treatment using before and after treatment data on Renal Calculi and Symptoms.

The level of significance probability 0.05 was used to test the treatment difference and the values are statistically significant.

Mean and Standard deviation of Renal calculi at before and after treatment

variable	Mean	Std.Deviation	Std.Error Mean	t VALUE	p VALUE
BTRLK	8.302	3.1685	.5010	6.092	P<0.001
ATRLK	4.200	4.0324	.6376		

The mean± standard deviation of renal calculi at before and after treatment were 8.30 ± 3.16 and 4.2 ± 4.03 respectively which is statistically significant (t= 6.092 p<0.001).

BTRLK – before treatment right left kidney

BTRLK – after treatment right left kidney

Mean and Standard deviation of clinical Symptoms before and after treatment

clinical Symptoms	Mean	Std.Deviation	Std.Error Mean	t VALUE	p VALUE
Before treatment	4.95	1.894	0.299	7.525	P < 0.0001
After treatment	2.93	0.656	0.104		

The mean± standard deviation of symptoms score at before and after treatment were 4.95 ± 1.89 and 2.93 ± 0.66 respectively which is statistically highly significant (t= 7.5 p<0.0001) i.e. the symptoms have been reduced significantly after the treatment.

USG ABDOMEN REPORT BEFORE AND AFTER TREATMENT									
S. NO	OPD/ IPD NO	AGE/ SEX			SIZE OF THE KIDNEY	SITE OF THE CALCULUS	NO OF CALCULUS	SIZE OF THE CALCULUS	HYDROURETERO NEPHROSIS
1	C69642	24/F	RT.KIDNEY	BT	9.7 ×4.4cm	lower calyx	2	3.2, 4.3mm	nil
				AT	105 ×42mm	**normal study**			nil
			LT.KIDNEY	BT	11.0× 5.1cm	-	-	-	nil
				AT	112× 40mm	-	-	-	nil
2	C71045	31/M	RT.KIDNEY	BT	10.5× 4.7cm	lower pole	1	4.8mm	nil
				AT	10.9× 4.4cm	**normal study**			nil
			LT.KIDNEY	BT	10.1× 4.5cm	-	-	-	nil
				AT	10.3 ×5.2cm	-	-	-	nil
3	C73030	43/M	RT.KIDNEY	BT	91.3 ×61.4mm	upper, middle calyx	3	3.0, 5.5mm, 4.5mm	nil
				AT	10.1 ×4.4cm	upper pole	1	5.1mm	nil
			LT.KIDNEY	BT	98.8 ×54.4mm	upper, middle calyx	3	3.0, 5.7mm, 3.0mm	nil
				AT	10.3× 5.2cm	lower pole	1	5.7mm	nil
4	C73050	43/M	RT.KIDNEY	BT	102.8× 53.6mm	mid, lower pole	4	5.0mm, 4.0,3.0mm	nil
				AT	102.6 × 53.6mm	mid, lower pole	3	3.5mm, 4.0,2.5mm	nil
			LT.KIDNEY	BT	87.5 × 53.7mm	upper, mid, lower pole	4	4.0,3.0mm, 6.0,3.0mm	nil
				AT	87.5× 53.7mm	upper, mid pole	3	4.0mm, 6.0,4.0mm	nil
5	C72496	32/F	RT.KIDNEY	BT	9.4 ×2.9cm	lower pole	1	4.0 mm	nil
				AT	9.0× 4.1cm	**normal study**			nil
			LT.KIDNEY	BT	9.4× 4.8cm	-	-	-	nil
				AT	9.5× 4.7cm	-	-	-	nil
6	C74152	52/M	RT.KIDNEY	BT	90.9× 43.3mm	inter polar calyx	2	8.9, 8.3mm	nil
				AT	89.6× 36.7mm	**normal study**			nil
			LT.KIDNEY	BT	101.3× 53.4mm	pelvic calyceal system	multiple	9.5mm	nil
				AT	99.5 × 57.6mm	**normal study**			nil

S.#	OPD/ IPD NO	AGE/ SEX			SIZE OF THE KIDNEY	SITE OF THE CALCULUS	NO OF CALCULUS	SIZE OF THE CALCULUS	HYDROURETERONEPHROSIS
7	C73299	32/M	RT.KIDNEY	BT	100.9× 44.2mm	-	-	-	nil
				AT	104.0 ×44.5mm	-	-	-	nil
			LT.KIDNEY	BT	107.6× 52.5mm	mid calyx	1	5.6mm	nil
				AT	111.2× 55.omm	mid calyx	1	5.7mm	nil
8	C73332	42/F	RT.KIDNEY	BT	96 ×43mm	lower pole	multiple	4mm	nil
				AT	10.7 ×4.7cm	lower pole	3	3mm	nil
			LT.KIDNEY	BT	92 ×49mm	mid pole	multiple	3.5mm	nil
				AT	10.7 ×5.0cm	upper, inter, lower pole	3	3-4mm	nil
9	C75054	50/M	RT.KIDNEY	BT	1o.2cm long	-	-	-	nil
				AT	1o.2cm long	-	-	-	nil
			LT.KIDNEY	BT	11.0cm long	mid calyx, mid ureter	2	8mm, 12mm	+
				AT	10.5cm long	mid calyx	1	8mm	nil
10	C76543	24/M	RT.KIDNEY	BT	10cm long	upper pole	1	6-7mm	nil
				AT	102× 46.6mm	**normal study**			nil
			LT.KIDNEY	BT	10.5cm long	lower pole	1	3-4mm	nil
				AT	97.8 ×53.2mm	**normal study**			nil
11	C74751	33/M	RT.KIDNEY	BT	95.9 ×50.6mm	middle calyx	few	4.8mm	nil
				AT	10.3 ×4.8cm	upper, mid calyx	2	0.3,0.8 ×0.5mm	nil
			LT.KIDNEY	BT	97.3 ×55.3mm	middle calyx	1	4.2mm	nil
				AT	10.3 ×4.9cm	**normal study**			nil
12	C77242	34/M	RT.KIDNEY	BT	9.6×4.3cm	inter polar calyx	2	6.4mm, 7.2mm	nil
				AT	10.0×4.8cm	inter polar calyx	1	6.9mm	nil
			LT.KIDNEY	BT	10.1 ×5.3cm	-	-	-	nil
				AT	10.1 ×4.9cm	-	-	-	nil
13	C77701	28/F	RT.KIDNEY	BT	12.2× 5.4cm	dilated system	few	8-10mm	+
				AT	12.2× 5.4cm	dilated system	few	8-10mm	+
			LT.KIDNEY	BT	11.3× 5.4cm	-	-	-	nil

S. NO	OPD/ IPD NO	AGE/ SEX			SIZE OF THE KIDNEY	SITE OF THE CALCULUS	NO OF CALCULUS	SIZE OF THE CALCULUS	HYDROURETERONEPHROSIS
				AT	11.3 ×5.0cm	-	-	-	nil
14	C78713	53/M	RT.KIDNEY	BT	9.2 ×4.4cm	mid pole	1	5.0mm	nil
				AT	8.6 ×4.2cm	**normal study**			nil
			LT.KIDNEY	BT	9.2× 4.6	lower pole	1	3.0mm	nil
				AT	8.8× 4.8cm	**normal study**			nil
15	C79434	29/M	RT.KIDNEY	BT	8.9× 3.9cm	upper calyx	1	5mm	nil
				AT	8.3× 3.5cm	upper calyx	1	4mm	nil
			LT.KIDNEY	BT	9.5× 3.8cm	-	-	-	nil
				AT	9.1× 4.0cm	-	-	-	nil
16	C78007	38/M	RT.KIDNEY	BT	11.0× 6.0cm	mid ureter	1	10mm	+
				AT	11.8 ×6.2cm	mid ureter	1	8mm	+
			LT.KIDNEY	BT	11.0 ×5.5cm	-	-	-	nil
				AT	10.3× 5.3cm	-	-	-	nil
17	C80489	40/F	RT.KIDNEY	BT	10.0× 4.cm	-	-	-	nil
				AT	92.9× 44.1mm	middle calyx	1	4mm	nil
			LT.KIDNEY	BT	11.0 ×5.0cm	VUJ	1	0.9,0.6cm	+
				AT	97.5× 54.0mm	**normal study**			nil
18	C80919	29/M	RT.KIDNEY	BT	9.6 ×4.3cm	upper, lower pole	2	5.4, 11.4×8mm	nil
				AT	9.4 ×4.5cm	lower pole	1	11.4×7.7mm	nil
			LT.KIDNEY	BT	11.4× 5.5cm	-	-	-	nil
				AT	11.2× 5.8cm	-	-	-	nil
19	C81353	60/M	RT.KIDNEY	BT	117.0 ×51.7mm	mid ureter	1	7.5mm	+
				AT		**normal study**			nil
			LT.KIDNEY	BT	104.8 ×55.4mm	mid pole	1	2.5mm	nil
				AT		**normal study**			nil
20	C81148	36/M	RT.KIDNEY	BT	9.9× 5.4cm	upper, inter, lower pole	multiple	6.0mm	nil
				AT	1o.4× 6.2cm	lower pole	multiple	5.0mm	nil
			LT.KIDNEY	BT	9.1×5.2cm	inter, lower pole	multiple	5.6mm	nil
				AT	10.1 ×5.5cm	mid pole	multiple	3.0mm	nil
21	C82243	22/M	RT.KIDNEY	BT	9.3× 3.9cm	-	-	-	nil
				AT	9.4 ×3.8cm	-	-	-	nil

S. NO	OPD/ IPD NO	AGE/ SEX			SIZE OF THE KIDNEY	SITE OF THE CALCULUS	NO OF CALCULUS	SIZE OF THE CALCULUS	HYDROURETERONEPHROSIS
			LT.KIDNEY	BT	10.2× 3.7cm	lower pole	1	4.5mm	nil
				AT	10.0× 4.4cm	**normal study**			nil
22	C81712	60/M	RT.KIDNEY	BT	11.6 ×5.0cm	proximal ureter	1	10-15mm	+
				AT	9.8× 5.7cm	lower ureter	1	8mm	+
			LT.KIDNEY	BT	10.7× 5.0cm	mid pole	1	5mm	+
				AT	10.2× 5.4cm	mid pole	1	4mm	+
23	C82707	42/M	RT.KIDNEY	BT	103.4× 45.7mm	-	-	-	nil
				AT	109.2 ×43.6mm	-	-	-	nil
			LT.KIDNEY	BT	116.6 ×61.4mm	VUJ	1	6.6mm	+
				AT	117.1 ×50.6mm	**normal study**			nil
24	C82990	27/M	RT.KIDNEY	BT	8.5× 3.7cm	upper, mid pole	multiple	5.0, 4.0mm	+
				AT	8.5 ×3.7cm	upper, mid pole	microliths		nil
			LT.KIDNEY	BT	9.0× 4.7cm	lower pole	1	5.0mm	nil
				AT	9.0× 4.7cm	lower pole	microliths		nil
25	C84030	38/M	RT.KIDNEY	BT	8.0× 3.7cm	-	-	-	nil
				AT	80.7× 34.9mm	-	-	-	nil
			LT.KIDNEY	BT	9.6× 6.2cm	proximal ureter	1	10mm	+
				AT	101.4× 56.7mm	**normal study**			nil
26	C80753	48/M	RT.KIDNEY	BT	100.1 ×45.2mm	lower calyx	1	4.2mm	nil
				AT	92.4 ×48.1mm	**normal study**			nil
			LT.KIDNEY	BT	84.1 ×96.6mm	lower pole	microliths		+
				AT	90.4 ×49.1mm	**normal study**			nil
27	C85766	32/M	RT.KIDNEY	BT	103.8 ×49.8mm	upper calyx	1	4.4mm	nil
				AT	98.4 ×47.9mm	**normal study**			nil
			LT.KIDNEY	BT	100.8× 46.7mm	-	-	-	nil
				AT	96.3 ×46mm	-	-	-	nil
28	C85523	25/F	RT.KIDNEY	BT	10.6 ×4.3cm		multiple	4mm	+
				AT	9.9 ×4.0cm		multiple	5-6mm	+

S. NO	OPD/ IPD NO	AGE/ SEX			SIZE OF THE KIDNEY	SITE OF THE CALCULUS	NO OF CALCULUS	SIZE OF THE CALCULUS	HYDROURETERONEPHROSIS
			LT.KIDNEY	BT	11.4× 5.5cm		multiple	5-6mm	+
				AT	10.8× 4.7cm		multiple	5-6mm	+
29	C87868	42/M	RT.KIDNEY	BT	9.9× 4.1cm	-	-	-	nil
				AT	1o.0× 4.1cm	mid pole	1	microlith	nil
			LT.KIDNEY	BT	10.1 ×4.3cm	mid pole	1	6.0mm	nil
				AT	10.2 ×4.3cm	mid pole	1	microlith	nil
30	C86025	40/M	RT.KIDNEY	BT	10.2× 4.9cm	upper pole	multiple	5.2mm	nil
				AT	10.5× 4.8	upper pole	multiple	<5.0mm	nil
			LT.KIDNEY	BT	9.4× 5.5cm	mid pole	multiple	5.5mm	nil
				AT	10.3× 5.2	mid pole	multiple	<5.0mm	nil
31	C88682	45/F	RT.KIDNEY	BT	9.2× 4.2cm	upper pole	multiple	5.7mm	nil
				AT	9.1× 4.0cm	upper pole	multiple	5.2mm	nil
			LT.KIDNEY	BT	10.2 ×4.2cm	lower pole	multiple	4.9mm	nil
				AT	9.2 ×4.2cm	lower pole	multiple	4.0mm	nil
32	C89476	33/F	RT.KIDNEY	BT	9.6× 3.7cm	mid calyx	1	0.5cm	nil
				AT	9.9 ×3.9cm	**normal study**			nil
			LT.KIDNEY	BT	10.0× 4.0cm	lower calyx	3	0.4, 0.5, 1cm	nil
				AT	10.2 ×5.2cm	**normal study**			nil
33	C95276	37/F	RT.KIDNEY	BT	10.3 ×3.7cm	-	-	-	nil
				AT	10.1× 3.9cm	-	-	-	nil
			LT.KIDNEY	BT	10.7 ×4.7cm	mid calyx	1	4mm	nil
				AT	11.1× 4.8cm	**normal study**			nil
34	3888	52/M	RT.KIDNEY	BT	102.0 ×50.0mm	middle calyx	2	6.0, 4.0mm	nil
				AT	9.2× 4.4cm	inter polar region	few	6mm	nil
			LT.KIDNEY	BT	100.2× 49.0mm	middle calyx	2	4.0, 3.0mm	nil
				AT	9.4× 5.3cm	inter polar region	microliths		nil
35	4965	32/M	RT.KIDNEY	BT	8.5 ×4.3cm	upper,middle lower calyx	multiple	3.0, 5.0mm	nil
				AT	109.7 ×44.2mm	upper,middle lower calyx	multiple	6mm	nil
			LT.KIDNEY	BT	8.6 ×4.5cm	upper,middle lower calyx	multiple	3.0, 5.0mm	nil
				AT	99.5 ×64.7mm	middle calyx	1	4.0mm	nil

S. NO	OPD/ IPD NO	AGE/ SEX			SIZE OF THE KIDNEY	SITE OF THE CALCULUS	NO OF CALCULUS	SIZE OF THE CALCULUS	HYDROURETERONEPHROSIS
36	5055	48/F	RT.KIDNEY	BT	92.4× 50.0mm	-	-	-	nil
				AT	98.8× 37.0mm	-	-	-	nil
			LT.KIDNEY	BT	94.2× 50.4mm	inter polar region	2	3.8, 4.3mm	nil
				AT	101.1× 41.9mm	lower calyx	1	4.1mm	nil
37	4061	32/F	RT.KIDNEY	BT	89× 40mm	upper pole	2	3mm, 4mm	nil
				AT	10.9× 4.5cm	lower pole	1	5mm	nil
			LT.KIDNEY	BT	93 ×43mm	lower pole	1	4mm	nil
				AT	11.5× 4.9cm	inter polar region	1	5mm	nil
38	4099	35/F	RT.KIDNEY	BT	10.1× 4.6cm	-	-	-	nil
				AT	10.1× 4.6cm	-	-	-	nil
			LT.KIDNEY	BT	11.5× 4.7cm	lower calyx	1	6mm	nil
				AT	11.6× 4.6cm	lower calyx	1	4mm	nil
39	4156	55/M	RT.KIDNEY	BT	9.2× 3.2cm	upper pole	1	2-4mm	nil
				AT	8.8 ×3.8cm	upper pole	1	2-4mm	nil
			LT.KIDNEY	BT	9.0× 3.6cm	upper pole	1	2-4mm	nil
				AT	8.9× 3.5cm	upper pole	1	2-4mm	nil
40	5115	27/M	RT.KIDNEY	BT	97 5×3.1mm	middle calyx, lower ureter	2	7.5, 8.5mm	+
				AT	96.2× 3mm	middle calyx	1	5.1mm	+
			LT.KIDNEY	BT	99.3× 58mm	-	-	-	nil
				AT	99×56mm	-	-	-	nil

BT – Before Treatment

AT – After Treatment

RENAL CALCULUS

OP No: C84030 Age: 38/M Stone size: 5mm

OP No: C85523 Age: 25 /M Stone size: 5mm

OP No: C73332 Age: 42 /M

Stone size: 3.5mm

OP No: C77242 Age: 34/M

Stone size: 5mm

DISCUSSION

The main aim of the treatment was to evaluate the therapeutic effect of the drug **Karpoora silasathu parpam** (Internal) in the disease **Azhal kalladaippu**. The clinical features of Azhal kalladaippu can be correlated to Renal Calculus in Modern science. As per yougi vaithiya chinthamani text, Azhal kalladaippu is characterized by oliguria, urethral pain mimics a pain caused by an insertion of burning iron bar in the urethra, sweating all over body, anuria, agonizing pain, blood stained calculus stagnated in urethra.

- The safety of the trial drug usage and standardization of the trial drug through biochemical analysis were also ensured during the study.
- The drug was prepared in the Gunapadam lab of National Institute of Siddha after the authentication of the raw drugs by the concerned department. The trial drug was prepared by the standard operating procedure as mentioned in the protocol.
- The preclinical toxicity studies (Acute and sub acute toxicity) for the above said trial drug was conducted at National Institute of Siddha after getting the proper acceptance and permission from the Institutional Animal Ethical Committee (IAEC). The trial drug was proved to be safe for human beings from the observations made from the study.
- The biochemical qualitative and quantitative analysis were done at the biochemistry lab of NIS and IIT Chennai respectively. It revealed the presence of effective minerals and the existence of the drug molecules at micro level.
- The clinical study was conducted with a well defined protocol and a proper proforma after the approval of the Institutional Ethical Committee (IEC). After screening 60 cases reporting at the OPD of department of Maruthuvam, 40 cases were selected for induction to the trial. Before enrollment into the trial the informed consent was obtained from the patients.
- The patients were treated for a period of 48 days with karpoora silasathu parpam (Internal medicine) at the dose of 130 mg, twice a day with the adjuvant of radish juice.

- Clinical assessment was done during each visit in OPD patients (7 days once) and daily for IPD patients and the data were noted in the prescribed proforma.
- Laboratory investigations & USG Abdomen were done on the 0day, & 48th day of the trial for both OP & IP patients. For IP patients, who was not in a situation to stay in the hospital for a long time was advised to attend the OPD for the continuation of the treatment. All the patients were put under observation for 2 months follow up period without the trial drug treatment.

THE OBSERVATIONS DISCUSSED BELOW:

GENDER DISTRIBUTION

The majority affected sex was male i.e, 28 cases (70%) and female was 12cases (30%).

Inference:

- Testosterone may cause increased oxalate production in men.
- Women have higher urinary citrate concentrations.

AGE DISTRIBUTION

This study showed that the highest incidence of Azhal kalladaippu was between 31 -40 years of age, i.e. 16 cases (40%).

Inference:

The peak incidence of renal calculi occurs between 20 and 40 years of age. As per the Rathina surukka naadi these period is mainly in pitha kaalam (33 – 66 years) of human life.

GUNAM DISTRIBUTION

All the 40 cases under this analysis were predominantly of Rajo gunam assessed from interrogation and other observations.

KAALAM DISTRIBUTION

Among the 40 patients, 25 cases (62.5%) were found to be in pitha kaalam (34-66 yrs) and 15cases (37.5%) in vatha kaalam (upto 33 years).

DIET

Among the 40 cases 38 cases (95%) were non-vegetarians (mainly animal protein and fat substances).

Inference:

Animal protein contains oxalates, calcium, phosphates and other elements often lead to an excess excretion of them in urine. High intake of animal protein causes high urinary oxalate, low pH, low urinary citrate, and High salt intake causes hypercalciuria. However, a reduced calcium diet can increase the risks of further stone formation.

DISTRIBUTION OF CASES BY PARUVAKAALAM (SEASONS)

In this study, 27 cases (67.5%) were reported in muthuvenil kaalam, 9cases (22.5%) in Elavenil kaalam and 4cases (10%) in Kaar kaalam. In Muthuvenil kaalam vatham is vitiated and Iyam comes to normal. In Elavenil kaalam Iyyam is vitiated.

Inference:

Environmental temperature with stone formation being more common in the summer months. This tendency may be due to relative dehydration and the subsequent production of concentrated, acidic urine. Alternatively some workers suggest the increased exposure to sunshine leads to increased urinary calcium excretion.

THINAI DISTRIBUTION

In this study, 90% of cases were reported from Neithal land.

Inference:

In Siddha literatures, it was mentioned that Neithal is a land, which is responsible for vaatha diseases. Mineral contents of water in this Neithal land may contribute to the formation of kidney stone.

OCCUPATIONAL REFERENCES

Sedentary work style accounts for the highest number of 13cases (32.5%).

Inference:

Sedentary occupations predispose to stones compared with manual workers. The risk of calcium oxalate and uric acid stones formation in Astronauts because of decreased pH and lower urinary volumes.

TREATMENT HISTORY

26cases (65%) had taken allopathic treatment in the past and had discontinued the same. The rest of the 14cases (35%) had not taken any other drugs prior to enrolling for the study.

DISTRIBUTION OF CASES BY CHRONICITY OF ILLNESS

Most of the cases (72.5%) were affected in the duration of upto 3 months. 10cases (25%) were affected by the illness from 4 to 6 months, rest of 1case (2.5%) was affected by the illness above 9 months.

CLINICAL FEATURES

Abdominal pain and pain from loin to groin region were present in almost all the 40 cases (100%). 32cases (80%) had yellow coloured urination. 27cases (67.5%) had oliguria and burning micturition. 10cases (25%) were affected by dysuria. 9cases (22.5%) were affected by agonizing pain. 8cases (20%) had pain in urethra. Only 4cases (10%) were nausea and vomiting.

DERANGEMENTS OF VATHAM

Viyanan and samanan were affected in all the 40 cases (100%) which resulted in abdominal pain and pain from loin to groin region. Abanan was affected in 31cases (77.5%) which resulted in burning micturition, dysuria and oliguria.

DERANGEMENT OF PITHAM

Saathaga piththam was affected in all the cases (100%) as a result the patients were unable to perform their routine duties. Anarpitham was affected in 4 cases (10%) which resulted in nausea and vomiting.

DERANGEMENT OF KABAM

Pothagam was affected in 4cases (10%) which resulted in nausea and vomiting.

DERANGEMENT OF KOSANGAL

Vingyanamayakosam was affected in all the 40 cases (100%) which resulted in abdominal pain and loin pain and 4cases (10%) was affected in Annamaya kosam which resulted in nausea and vomiting.

DISTRIBUTION OF CASES BY NAADI

Vali azhal naadi was felt in 21 cases (52.5 %), azhal vali naadi was felt in 18 cases (45 %) and azhal naadi was felt in 1 case (2.5 %).

DISTRIBUTION OF CASES BY NEERKURI

On examination by Envagai thervugal, the moothiram has been affected in all patients due to derangement of Vatham and pitham. Yellow coloured was observed in 32 cases (80%) and straw coloured was in 8cases (20%). The volume of urine was reduced the amount in 27 cases (67.5%), rest of 13 cases (32.5%) had norml urine volume. Enjal was found to be in 12 cases (30%) in the presence of bacterial infections. No other changes were observed in Manam, Edai and Nurai.

DISTRIBUTION OF CASES BY NEIKKURI

In 27cases (68 %) oil was spread slowly like pearl shape. In fastly spread, 10cases (25 %) had annular/ring and 3cases (7%) had serpentine like.

DISTRIBUTION OF CALCULUS IN URINARY SYSTEM

15cases (37.5%) had Bilateral renal calculi.12cases (30%) were affected by ureteric calculus. 7cases (17.5%) were affected by Right renal calculus. 6cases (15%) were affected by Left renal calculus.

URINE CULTURE AND SENSITIVITY

Among the 40 cases, 12cases (30%) had Bacterial infections in urine culture before treated with trial drug. Out of them 5cases (41.7%) were found to be no bacterial infections in urine culture after the completion of the trial drug treatmen and 7 cases (58.3%) had grown in culture.

HYDROURETRONEPHROSIS

Before treated with trial drug 12 cases (30%) had hydrouretronephrosis. Among them, 7cases (58.3%) were relieved from hydrouretronephrosis after the treatment with trial drug and 5 cases (41.7%) were not relieved.

OUTCOME

PRIMARY OUTCOME OBSERVATION

RESULT OF USG ABDOMEN AFTER TREATMENT

All the 40 patients were taken ultrasonography, after the completion of the trial drug treatment,

- Among the 40 cases stone completely dissolved in 15 cases (37.5%) [Ureteric calculus, Renal calculus + Ureteric calculus]
- Size and number of stone is reduced in 20 cases (50%) [Renal calculus].
- In 5 cases (12.5%) there was no change in size of stone but clinical symptoms were completely relieved.

Based on the above results,

87.5% showed Good results and **12.5%** showed poor results.

Good - Normal study (stone completely dissolved)
Reduced its number (more than 2 / multiple)
Reduced its size.

Poor - No change in size and increase in size.

SECONDARY OUTCOME OBSERVATION

RESULT OF CLINICAL FEATURES AFTER TREATMENT

Abdominal pain and loin pain were relieved in 38 cases (95%) other symptoms such as abdominal distension, pain in urethra, agonizing pain, dysuria, oliguria, yellow coloured urination, burning micturition, nausea and vomiting, haematuria were relieved in all the 40 cases (100%).

IMPROVEMENT

Among 40 cases 38 cases (95%) had clinically good improvement (symptoms completely relieved) after treatment with trial drug, 2 cases (5%) had moderate improvement (symptoms reduced).

LABORATORY INVESTIGATIONS

Laboratory investigation of Blood, Urine and Stools were done for all 40 cases.

HAEMOGLOBIN:

Among 40 cases,

- In 22 (55%) of cases there was improvement in HB after treatment ranging between maximum 3.5gms% and minimum 0.3gms%.
- 1 (2.5%) cases remained in the same HB level after treatment.
- In 17 (42.5%) of cases there was reduction in HB ranging between maximum 2.2gms% and minimum 0.2gms%.

In the Haematological investigations other than Hb, all the reports i.e. RBC, WBC, LFT, RFT and Blood Glucose were within the normal range before and after Treatment with the trial drug.

BIOCHEMICAL ANALYSIS:

- Qualitative analysis of Karpoora silasathu parpam done in NIS biochemical lab revealed that the trial drug contains minerals like Calcium, Sulfur, Silicate, Zinc, Phosphate, potassium etc.
- Quantitative analysis revealed that it contains chiefly,

  - 458.885 mg/L Calcium
  - 76.235 mg/L Sulfur
  - 19.957 mg/L Silicate
  - 23.745 mg/L Phosphate
  - 25.124 mg/L potassium

TOXICITY STUDY:

ACUTE ORAL TOXICITY STUDY

Acute toxicity studies done in National Institute of Siddha, as per WHO guide lines revealed the safety of the drug at the dose of 4.68mg/kg/bw as it did not exhibit any mortality in mice. In necropsy there were no abnormalities detected in the internal organs such as, Liver, Heart, Lungs, pancreas Spleen, Stomach, Intestine, Kidney, Urinary bladder and Uterus.

SUB ACUTE TOXICITY STUDY

Sub acute toxicity studies done in National Institute of Siddha, as per WHO guide lines did not exhibit any mortality in rats. Animal behavior, metabolic functions (food and water intake, defaecation, urination etc) did not reveal any abnormality. Blood investigation parameters and histopathological examination did not show any abnormal variations.

Hence it can be concluded from the study that up to maximum dose 46.8mg/kg/bw (10X) the drug was proved to be safe.

SUMMARY

- The aim of the study was to evaluate the therapeutic efficacy of the drug karpoora silasathu parpam (Internal) in Azhal kalladaippu.
- Before initiating the clinical trial, approval was got from the Institutional Animal Ethical Committee (1248/ac/09/CPCSEA/4/04/2011 – 20/12/2011) and Institutional Ethical Committee (NIS/IEC/2011/3/04 – 24/12/2011) for conducting the pre clinical studies and clinical studies respectively by submitting the well defined protocol and proforma.
- The raw drugs were authenticated by the concerned department and the trial drug was prepared by the investigator in the Gunapadam lab of National Institute of Siddha as per the Standard Operating Procedure mentioned in the protocol.
- The medicine was then subjected to pre clinical toxicity studies (Acute and sub acute toxicity studies) as per the protocol and the safety of the drug was ensured.
- From the Acute oral toxicity study, the trial drug was found to be safe even at higher dose level of 4.68mg/kg/bw.
- From the sub acute toxicity study the trial drug at the dose of 46.8mg/kg/bw (10X) did not exhibit any mortality in rats.
- The qualitative and quantitative bio chemical studies were done at the bio chemistry lab of National Institute of Siddha and IIT Chennai respectively.
- The biochemical study of the trial drug reveals the presence of sulphate, potassium, phosphate and carbonate.
- Among the 60 cases screened at the OPD of department of Maruthuvam NIS, 40 cases were recruited for the trial as per the inclusion and exclusion criteria.
- Clinical diagnosis of Azhal kalladaippu was made by Siddha and Modern methodology.

- Before inducement into the trial informed consent was obtained from the patients. Out of the 40 cases 33 cases were treated in OPD and 7 cases in IPD.
- The patients were treated for a period of 48 days The trial medicine selected for Internal treatment were karpoora silasathu parpam (internal medicine) at the dose of 130 mg twice a day with adjuvant of Radish juice referred under Siddha literature Agathiyar chendhooram - 300, moolamum uraiyum 1st edition respectively.
- Required lab investigations were carried out before and after the treatment and the concerned data was recorded in the proforma.
- Clinical assessment was done daily in all the IP patients and in OP patients it was assessed once in 7 days.
- During the study period, there was no event of any adverse reactions owing to the drug or disease.
- Statistical analysis showed significant difference between before and after treatment in the kidney stone size ($p < 0.001$) and symptoms ($p < 0.0001$).
- 38 cases (95%) had clinically good improvement (symptoms completely relieved) after treatment with trial drug, 2 cases (5%) had moderate improvement (symptoms reduced).

All the 40 patients were taken ultrasonography, after the completion of the trial drug treatment,

- **87.5%** showed Good results and **12.5%** showed poor results.

Good - Normal study (stone completely dissolved)
Reduced its number (more than 2 / multiple)
Reduced its size.

Poor - No change in size and increase in size.

CONCLUSION

- The aim of the study was to evaluate the therapeutic efficacy of the drug karpoora silasathu parpam (Internal) in Azhal kalladaippu.
- The safety studies (Acute toxicity and sub acute toxicity) studies conducted revealed that the trial drug was safe even at higher dosage of 46.8 mg/animal. There were no abnormalities found in blood investigation and histopathological examination. Hence it can be reasonably assumed that the drug is safe for human.

Clinical study revealed the therapeutic efficacy of the trial drug by showing,

In the result of USG ABDOMEN,

- **87.5%** showed Good results and **12.5%** showed poor results.
- 38 cases (95%) had clinically good improvement 2 cases (5%) had moderate improvement in clinical symptoms.
- There were no adverse reactions complained during the trial.
- Statistical analysis showed significant difference between before and after treatment in the kidney stone size ($p < 0.001$) and symptoms ($p < 0.0001$).
- Because of the encouraging clinical outcome, the study may be further carried out with the same drug in a large number of cases.

TOXICOLOGICAL EVALUVATION OF KARPOORA SILASATHU PARPAM

ACUTE TOXICITY STUDY OF KARPOORA SILASATHU PARPAM

[WHO guidelines, 1993]

Principle:

Acute toxicity was carried out in Swiss albino mice with a single exposure of 10 times of the recommended therapeutic dose of test compound the study duration will be 14 days.

Animal species **:** Swiss albino mice

Age / Weight / Size **:** 6 weeks. Mice-20-25gms.

Gender **:** Both male and female

Number of Animals **:** Mice: 20

Acclimatization Period **:** 7 Days

Clinical dose **:** 260 mg\day

S.No	Group	No of mice
1	Vehicle control (radish juice)	10 (5 male, 5 female)
2	Toxic dose 10X therapeutic dose (4.68mg)	10 (5 male, 5 female)

Test Animals

Test animals were obtained from the animal laboratory of the King institute, Chennai and stocked at National institute of siddha, Chennai. All the animals were kept under standard environmental condition (27+ or – 2 degree c).The animals had free access to water and standard pellet diet (Sai Durga foods pvt.ltd, Bangalore).The principles of laboratory animal care were followed and the Institutional ethical committee approved the use of animals and the study design. **(1248/ac/09/CPCSEA/December/ 2011)**

Route of administration:

Oral route was selected, because it is the normal route of clinical administration.

Test substance and vehicle

The Karpoora silasathu parpam is ash in colour. The test substance is insoluble in water, in order to obtain and ensure the uniformity in drug distribution the drug is dissolved by aqueous Tween 80 solution (10%).

Administration of doses

Karpoora silasathu parpam was suspended in aqueous Tween 80 solution (10%), with uniform mixing and it was administered to the groups in a single oral dose .The control groups were received equal volume of the vehicle. The animals were weighed before giving the drug. The dose level was calculated according to body weight, and surface area. Since the clinical dose was 260mg\day it was converted to animal dose (4.68mg) and then administered. The principle of laboratory animal care was followed.

Observations

Observations were made and recorded systematically and continuously observed as per the guideline after substance administration. Animals were observed individually (visual observations included skin changes, alertness, grooming, aggressiveness, sensitivity to sound, touch and pain ,restlessness, tremors, convulsion, righting reflex, corneal reflex, gripping reflex, pinna reflex, writhing reflex, papillary reflex, urination, salivation, lacrimation for first 4 hrs, then periodically during the first 24 hrs. Animals were observed for body weight and mortality for 14 days. If animals dying during the period of study, the animals were sacrificed. At the end of the 14^{th} day all animals were sacrificed and necropsy was done.

Body Weight

Individual weight of animals was determined before the test substance was administered and daily for 14 days. Weight changes were calculated and recorded. At the end of the test, surving animals were weighed and sacrificed.

Results: karpoora silasathu param at the dose 4.68mg/animal did not exhibit any mortality in mice.

No behavior changes were noted for the first 4 hours and for the next 24 hours and throughout the study period of 14 days. No weight reduction was noted before and after the acute study duration. Reflexes were found to be normal before and after the study. All other observations were found to be normal before and after the study. In Necropsy, the organs of the animal such as, Liver, Heart, Lungs, Pancreas, Spleen, Stomach, Intestine, Kidney, Urinary bladder, Uterus all appeared normal.

SUB ACUTE TOXICITY STUDY OF KARPOORA SILASATHU PARPAM

Animals	**:**	Male and Female Wister albino rats
Age	**:**	6-8 weeks
Weight	**:**	150-200 gms
Gender	**:**	Both male and female
Number of animals	**:**	Rat: 40
Acclimatization period	**:**	7 Days
Clinical dose	**:**	260mg\day
Clinical duration	**:**	28 days

S.No	Group	No of Rats
1	Vehicle control (radish juice)	10 (5male,5 female)
2	1XTherapeutic dose (4.68 mg)	10 (5male,5 female)
3	5XTherapeutic dose (23.4mg)	10 (5male,5 female)
4	10XTherapeutic dose(46.8mg)	10(5male, 5 female)

Animal source:

Test animals were obtained from the animal laboratory of the King institute, Chennai, and stocked at national institute of siddha, chennai. All the animals were kept under standard environmental condition (27+ or – 2 degree c) .The animals had free access to water and standard pellet diet (Sai durga foods pvt.ltd, Bangalore). The principles of laboratory animal care were followed and the Institutional ethical committee approved the use of animals and the study design. **(1248/ac/09/CPCSEA/December/2011)**

Identification of animal:

By cage number, animal number and individual marking on fur.

Housing and Environment:

The animals were housed in polypropylene cages provided with bedding of husk. Dark and light cycle each of 12 hours.

Administration period:

The period of administration of the test substance to animals are depending on the expected period of clinical use. Since the clinical duration of the test drug is 28 days and as per WHO guidelines the administration period is reported to be 1 month.

Dose selection:

The results of acute toxicity studies in Swiss albino mice indicated that karpoora silasathu parpam was non toxic and no behavioral changes, mortality was observed. On the basis of these results, the doses were selected for the study as per WHO guidelines.

Preparation and administration of dose:

Karpoora silasathu parpam was suspended in aqueous twin 80 solution (10%). It was administered to animals at dose levels of 1X therapeutic dose (4.68mg/animal), 5X Therapeutic dose (23.4mg/animal) and 10X Therapeutic dose (46.8mg/animal).The control animals were administered vehicle only. Administration was by oral (gavage) once a day for 30 days.

METHODOLODY:

Randomization, numbering and grouping of animal:

The animals were randomly divided into three groups for dosing up to 30 days. Each group consist of 10 animals (5 per sex in each group) were allowed acclimatization period of 7 days to laboratory conditions prior to the initiation of treatment. Each animal fur was marked with picric acid. The females were nulliparous and non pregnant.

OBSERVATION:

Experimental animals were kept under observation throughout the course of study for the following

Body weight:

Weight of each rat was recorded on day 1 and at weekly intervals throughout the course of study and at termination to calculate relative organ weights. From the data mean body weights and percent body gain were calculated.

Food and water consumption:

The quantity of food consumed by groups consisting of an animal for different doses was recorded at weekly intervals. Food consumed per animal was calculated for control and the treated dose groups

Clinical sings

All animals were observed daily for clinical sings. The time of onset intensity and duration of this symptom if any were recorded.

Mortality:

All animals were observed twice daily for mortality during entire course of study.

TERMINAL STUDIES:

LABORATORY INVESTIGATIONS:

Following laboratory investigations were carried out. On day 31 animals fasted overnight. Blood samples were collected by cardiac puncture using sodium heparin (200IU\ml) for blood chemistry and potassium EDTA (1.5 mg/ml) for hematology anticoagulant. Blood sample were centrifuged at 3000 r. p .m for 10 minutes.

BIOCHEMICAL INVESTIGATIONS:

The effect of **karpoora silasathu parpam** on certain biochemical parameters were examined and compared with those of the control group. The blood samples collected with heparinized bottles were centrifuged at 5000 rpm for 10 minutes to obtain clear serum for the following investigation. Glucose was estimated using commercial Glucose estimation kit (Span Diagnostics) by the method of Barham *et al.,* (1972) and Tenscher. *et al.,* (1971),Haemoglobin PCV, RBC, Erythrocyte count was estimated by Hemocytometer method of Ghai (1995). Total Leukocyte Count was estimated by Hemocytometer method of John (1972).Total, (Bilirubin test kid-malloy and evelyn 1937) direct and indirect bilirubins were determined. Alkaline phosphatase, Alanine amino tranferase (ALT) and Aspartate amino transferase (AST) were measured by using ALT and AST test kit (kind & king) .Total protein TP concentration was determined. Albumin was determined based on its reaction with

bromocresol green (binding method). Urea was determined according to urease – berthelot method and plasma creatinine was estimated using jaffe reaction. Results of biochemical investigations conducted on day 31 revealed significant changes in the values of different parameters studied when compared with those of respective controls.

Statistical analysis done for the above said biochemical investigations did not reveal any significant difference in values between the control groups and test groups stating that the provided test drug is non toxic.

NECROPSY:

All the animals were sacrificed on day 31 under ether anesthesia. Necropsy of all animals was carried out and the weights of the organs including liver, kidneys, brain, heart, lungs and sex organs were recorded.

HISTOPATHOLOGY:

Tissue samples of organs from control and treated animals were preserved in 10% formalin for preparation of sections using microtome. The organs included brain, heart, lungs, stomach, liver, kidneys, spleen, intestine, pancreas and sex organs of the animals were preserved and they were subjected to histopathological examination.

The organ pieces (3-5 micron) were fixed in 10% formalin for 24 hours and washed in running water for 24 hours .Samples were dehydrated in tissue processor and then cleaned in benzene to remove absolute alcohol. Embedding was done by passing the cleared sample through three cups containing molten paraffin at 50 degree c and then a cubical block of paraffin made by the L moulds it was followed by microtome and the slides were stained with haematoxylin–eosin stain. Stained sections of each organ were examined under light microscope at high (40X) power magnification. All the histo pathological slides were prepared at Vels University, pallavaram, Chennai.

RESULTS:

CONTROL ANIMALS

Brain: Shows cerebral cortex with normal appearing glial cells.

Heart: Shows normal appearing myocardial fibres with patent coronaries.

Lung: Shows bronchioles, alveoli, widened alveolar septa and chronic inflammatory cells**.**

Stomach: Shows gastric mucosal glands lined by columnar cells.

Liver: Shows central veins with rows of radiating hepatocytes, Portal triads and cells appear normal.

Kidney: Shows glomeruli tubules, interstitial cells of normal histology

Spleen: Section of spleen shows congested red pulp and lymphoid follicles with germinal centres forming the white pulp

Intestine: Shows normal Intestinal mucosal lining with mild exudates.

Pancreas: Shows normal acini with islets of β-cells

Ovary: Shows ovarian stroma with follicles and corpus leuteum.

Testis: Shows normal tubules with spermatogenesis.

IMPRESSION: NORMAL STUDY

TEST GROUP ANIMALS

1X THERAPEUTIC DOSE

Brain: Shows normal brain with nerve fibers and astrocytes.

Heart: Shows normal cardiac muscle bundles.

Lung: Shows normal alveoli.

Stomach: Shows normal mucosal glands.

Liver: Shows almost normal hepatocytes and occasional binucleate cells.

Kidney: Shows normal renal tissue with glomeruli and tubules.

Spleen: Shows normal spleen with lymphoid aggregation.

Intestine: Shows normal Intestinal mucosal lining with mild exudates.

Pancreas: Shows normal acini with islets of β-cells

Ovary: Shows ovarian stroma with follicles and corpus leuteum.

Testis: Shows normal tubules with spermatogenesis.

IMPRESSION: NORMAL STUDY

5X THERAPEUTIC DOSE

Brain: Shows brain with edema, microglial proliferation, shows brain with micro cystic change and astrocytic proliferation, shows brain with mononuclear infiltrate around vessel.

Heart: Shows congestion and mild inflammatory infiltration in between cardiac muscle bundles.

Lung: Shows congested alveolar wall with mild thickening and mild emphysematous changes.

Stomach: Shows near normal mucosal gland with mild exudates.

Liver: Shows hepatocytes with focal mild fatty change.

Kidney: Shows renal tissue with focal tubular damage, interstitial inflammatory collection. Glomeruli show epithelial proliferation.

Spleen: Shows congestion with lymphoid hyperplasia.

Intestine: Shows normal Intestinal mucosal lining with mild exudates.

Pancreas: Shows pancreas with acini and normal islets.

Testis: Shows normal tubules with spermatogenesis.

Ovary: Shows ovarian stroma with follicles and corpus leuteum.

IMPRESSION: NORMAL STUDY

10X THERAPEUTIC DOSE

Brain: Shows brain with edema. Astrocytes show degenerative changes. Shows brain with pyknotic irregular nucleus, shows brain with vesicular nuclei and micro cystic changes.

Heart: Shows hypertrophic cardiac muscle bundles.

Lung: Shows congestion, narrowed alveolar space and thickened alveolar wall.

Stomach: Shows stomach with superficial erosion and congestion.

Liver: Shows marked dilatation of sinusoids, degeneration of hepatocytes, necrosis.

Kidney: Shows renal tissue with tubular epithelial damage.

Spleen: Shows lymphoid hyperplasia.

Intestine: Shows normal Intestinal mucosal lining with mild exudates.

Pancreas: Shows atrophic islet cells.

Testis: Giant cells were formed in the lumen of the seminiferous tubules and the spermatogenic cells degenerated.

Ovary: Shows ovarian follicles and corpus leuteum.

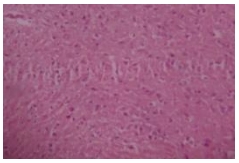
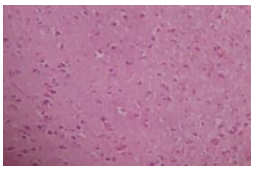
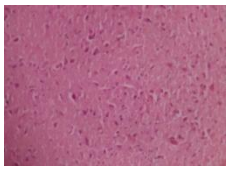
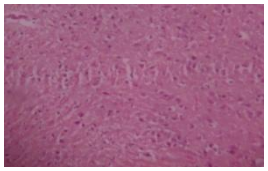
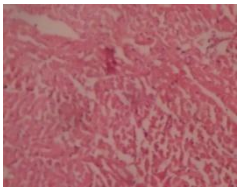
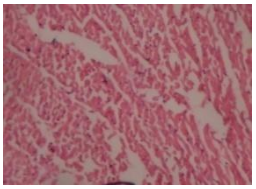
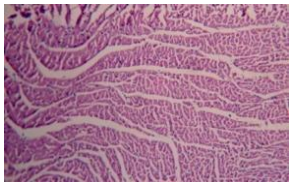
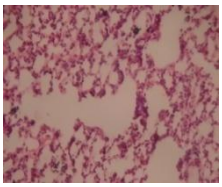
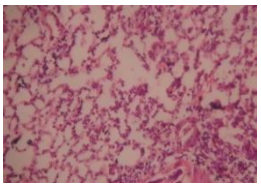
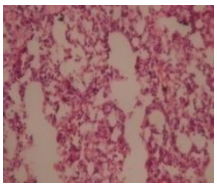
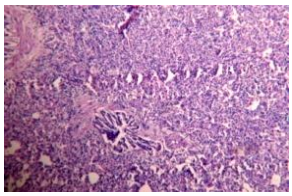
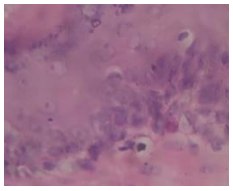
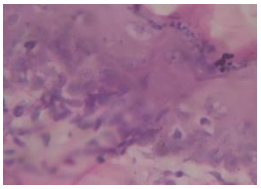
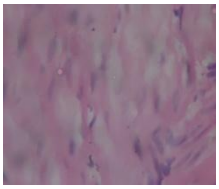
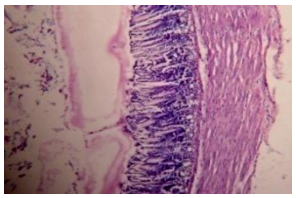
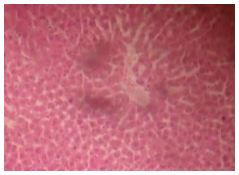
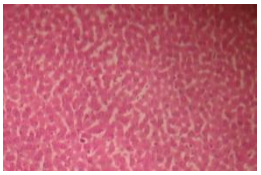
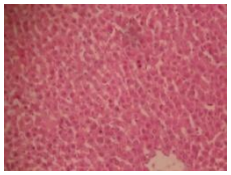
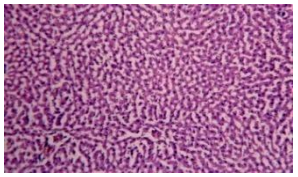
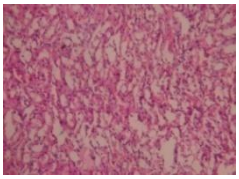
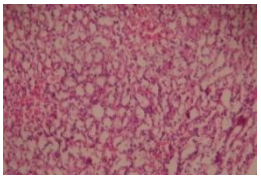
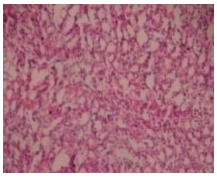
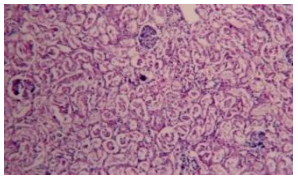
IMPRESSION: NORMAL STUDY

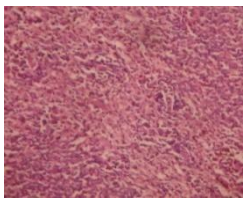
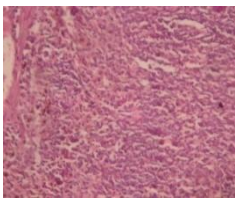
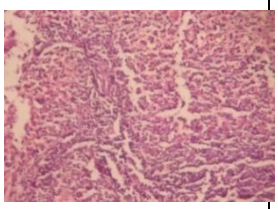
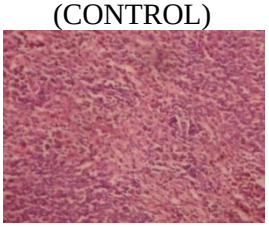
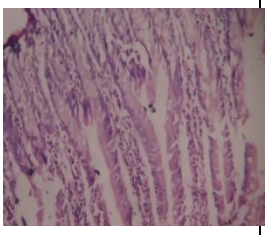
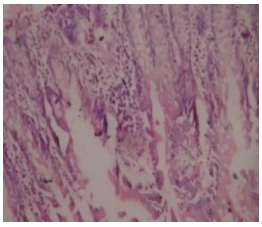
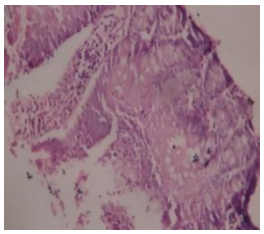
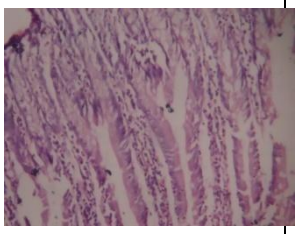
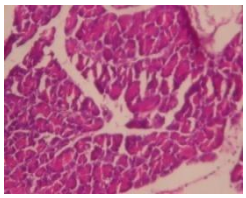
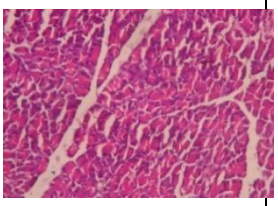
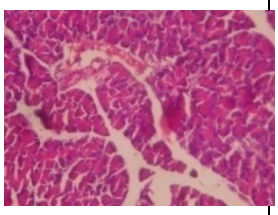
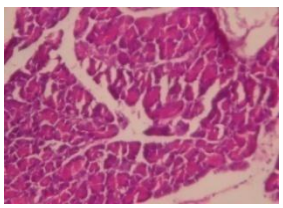
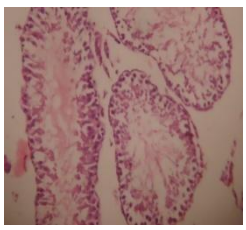
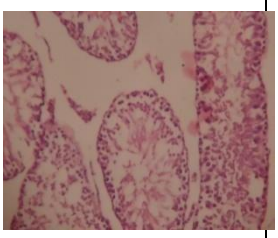
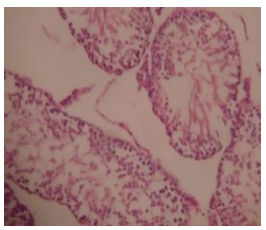
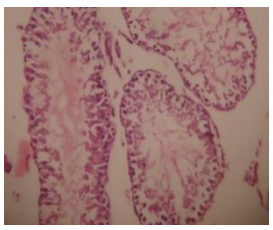
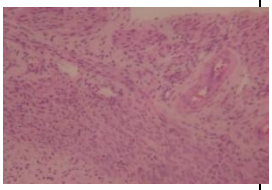
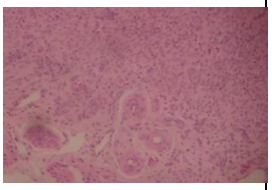
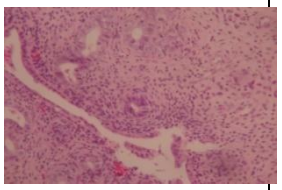
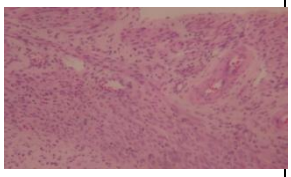
The histopathological studies did not reveal any abnormalities in the animals in both control and test groups stating that the drug is non toxic.

RESULTS:

- No weight loss, abnormal animal behaviours, metabolic functions [urination, lacrimation, defaecation etc.,] and mortality were noted.
- In necropsy of the animal organs showed normal appearance and weight.
- All Haematological and biochemical parameters were within normal limits.
- The stastical report of the Haematological and Biochemical datas didnot show any significant difference, between the control and test groups.
- In Histopathological studies, No abnormal findings were observed in the organs such as brain, heart, lungs, stomach, liver, kidneys, spleen, intestine, pancreas and sex organs in X, 5X and 10X compared with control group.

HISTOPATHOLOGY PHOTOS

BRAIN(X) 	BRAIN(5X) 	BRAIN10(X) 	BRAIN(CONTROL) 
HEART(X) 	HEART(5X) 	HEART(10X) 	HEART(CONTROL) 
LUNGS(X) 	LUNGS(5X) 	LUNGS(10X) 	LUNGS(CONTROL) 
STOMACH(X) 	STOMACH(5X) 	STOMACH(10X) 	STOMACH(CONTROL) 
LIVER(X) 	LIVER(5X) 	LIVER(10X) 	LIVER (CONTROL) 
KIDNEY(X) 	KIDNEY(5X) 	KIDNEY(10X) 	KIDNEY (CONTROL) 

SPLEEN(X) 	SPLEEN(5X) 	SPLEEN(10X) 	SPLEEN (CONTROL) 
INTESTINE(X) 	INTESTINE(5X) 	INTESTINE(10X) 	(INTESTINE (CONTROL) 
PANCREAS(X) 	PANCREAS(5X) 	PANCREAS(10X) 	PANCREAS(CONTROL) 
TESTIS(X) 	TESTIS(5X) 	TESTIS(10X) 	TESTIS(CONTROL) 
OVARY(X) 	OVARY(5X) 	OVARY(10X) 	OVARY (CONTROL) 

BIO -CHEMICAL ANALYSIS OF KARPOORA SILASATHU PARPAM

ANALYSED AT NATIONAL INSTITUTE OF SIDDHA

S.No	EXPERIMENT	OBSERVATION	INFERENCE
1.	Appearance of sample	Ash in colour	
2.	**Solubility:** a. A little (500mg) of the sample is shaken well with distilled water. b. A little (500mg) of the sample is shaken well with con. HCl/Con. H_2So_4	Sparingly soluble	Presence of Silicate
3.	**Action of Heat:** A small amount (500mg) of the sample is taken in a dry test tube and heated gently at first and then strong.	No White fumes evolved	Absence of Carbonate
4.	**Flame Test:** A small amount (500mg) of the sample is made into a paste with con. HCl in a watch glass and introduced into non-luminous part of the Bunsen flame.	No Bluish green flame appeared.	Absence of Copper
5.	**Ash Test:** A filter paper is soaked into a mixture of sample and dil. cobalt nitrate solution and introduced into the Bunsen flame and ignited	Yellow colour flame	Presence of Sodium

PREPARATION OF EXTRACT:

5gm of **Karpoora silasathu parpam** is weighed accurately and placed in a 250ml clean beaker and added with 50ml of distilled water. Then it is boiled well for about 10 minutes. Then it is cooled and filtered in a 100ml volumetric flask and made up to 100ml with distilled water.

S.No	EXPERIMENT	OBSERVATION	INFERENCE
	I. Test For Acid Radicals		
1.	**Test For Sulphate :** 2ml of the above prepared extract is taken in a test tube to this added 2ml of 4%dil. ammonium oxalate solution.	Cloudy appearance present	Presence of Sulphate
2.	**Test For Chloride:** 2ml of the above prepared extracts is added with 2ml of dil-HCl is added until the effervescence ceases off.	Cloudy appearance present.	Presence of Chloride
3.	**Test For Phosphate:** 2ml of the extract is treated with 2ml of ammonium molybdate solution and 2ml of con.HNO_3	Mild yellow appearance present	Presence of phosphate
4.	**Test For Carbonate:** 2ml of the extract is treated with 2mldil. magnesium sulphate solution	No of cloudy appearance	Absence of Carbonate
5.	**Test For Fluoride & Oxalate:** 2ml of extract is added with 2ml of dil. Acetic acid and 2ml dil.calcium chloride solution and heated.	Presence of cloudy appearance	Presence of fluoride and oxalate

6.	**Test For Nitrate:** 1gm of the substance is heated with copper turning and concentrated H2SO4 and viewed the test tube vertically down	No Brown gas is evolved	Absence of Nitrate
7.	**Test For Sulphide:** 1gm of the substance is treated with 2ml of con. HCL	Rotten Egg Smelling gas evolved	Presence of Sulphide
8.	**Test For Nitrite:** 3drops of the extract is placed on a filter paper, on that-2 drops of dil.acetic acid and 2 drops of dil. Benzidine solution is placed.	No Characteristic changes	Absence of Nitrite
9.	**Test For Borate:** 2 Pinches (50mg) of the substance is made into paste by using dil.sulphuric acid and alcohol (95%) and introduced into the blue flame.	Bluish green colour flame not appeared	Absence of Borate
II. TEST FOR BASIC RADICALS			
1.	**Test For Lead:** 2ml of the extract is added with 2ml of dil.potassium iodine solution.	Yellow Precipitate is obtained.	Presence of Lead
2.	**Test For Copper:** One pinch (50mg) of substance is made into paste with con. HClin a watch glass and introduced into the non-luminuous part of the flame.	No blue colour precipitate formed.	Absence of Copper

3.	**Test For Aluminium:** To the 2ml of extract dil.sodium hydroxide is added in 5 drops to excess.	No Characteristic changes	Absence of Aluminium
4.	**Test For Iron:** a. To the 2ml of extract add 2ml of thiocyanate ammonium solution b. To the 2ml of extract add 2ml of thiocyanate ammonium solution and 2ml of con HNO_3 .	Mild red colour appear	Presence of Iron
5.	**Test For Zinc:** To 2ml of the extract dil.sodium hydroxide solution is added in 5 drops to excess and dil.ammonium chloride is added.	White precipitate is not formed	Absence of Zinc
6.	**Test For Calcium:** 2ml of the extract is added with 2ml of 4% dil.ammonium oxalate solution	Cloudy appearance and white precipitate is obtained	Presence of Calcium
7.	**Test For Magnesium:** To 2ml of extract dil.sodium hydroxide solution is added in drops to excess.	No White precipitate is obtained	Absence of Magnesium
8.	**Test For Ammonium:** To 2ml of extract 1 ml of Nessler's reagent and excess of dil.sodium hydroxide solution are added.	No Brown colour appeared	Absence of Ammonium

9.	**Test For Potassium:** A pinch (25mg) of substance is treated off with 2ml of dil.sodium nitrite solution and then treated with 2ml of dil.cobalt nitrate in 30% dil.glacial acetic acid.	Yellowish Precipitate is obtained.	Presence of Potassium
10.	**Test For Sodium**: 2 pinches (50mg) of the substance is made into paste by using HCl and introduced into the blue flame of Bunsen burner.	Yellow colour flame appeared	Presence of Sodium
11.	**Test For Mercury**: 2ml of the extract is treated with 2ml of dil.sodium hydroxide solution.	No Yellow precipitate is obtained	Absence of Mercury
12.	**Test For Arsenic:** 2ml of the extract is treated with 2ml of dil.sodium hydroxide solution.	No brownish red precipitate is obtained	Absence of Arsenic
	III. MISCELLANEOUS		
1.	**Test For Starch:** 2ml of extract is treated with weak dil.iodine solution	No Blue colour developed	Absence of Starch
2.	**Test For Reducing Sugar:** 5ml of Benedict's qualitative solution is taken in a test tube and allowed to boil for 2 minutes and added 8 to 10 drops of the extract and again boil it for 2 minutes. The colour changes are noted	Brick red colour not developed	Absence of Reducing sugar

3.	**Test For The Alkaloids:** a) 2ml of the extract is treated with 2ml of dil.potassium lodide solution. b) 2ml of the extract is treated with 2ml of dil.picric acid. c) 2ml of the extract is treated with 2ml of dil.phosphotungstic acid.	Yellow colour developed	Absence of Alkaloid
4.	**Test For Tannic Acid:** 2ml of extract is treated with 2ml of dil.ferric chloride solution	No Black precipitate is obtained	Absence of Tannic acid
5.	**Test For Unsaturated Compound:** To the 2ml of extract 2ml of dil.Potassium permanganate solution is added.	Potassium permanganate is not decolourised	Absence of Unsaturated compound
6.	**Test For Amino Acid:** 2 drops of the extract is placed on a filter paper and dried well. 20ml of Biurette reagent is added.	Violet colour developed	Absence of Amino acids
7.	**Test For Type Of Compound:** 2ml of the extract is treated with 2 ml of dil.ferric chloride solution.	No Brown colour developed	Absence of Oay quinole, Pinephrine and Pyro catechol

PRELIMINARY QUALITATIVE PHYTOCHEMICAL TESTS PROCEDURE AND INTERPRETATION OF RESULTS

S.NO	PROCEDURE	INFERENCE
1.	**Calcium**	Presence of Calcium
2.	**Sulphate**	Presence of Sulphate
3.	**Chloride**	Presence of Chloride
4.	**Carbonate**	Absence of Carbonate
5.	**potassium**	Presence of potassium
6.	**Iron**	Presence of Iron
7.	**Phosphate**	Presence of Phosphate
8.	**Tannic acid**	Absence of Tannic acid
9.	**Aluminium**	Absence of Aluminium
10.	**Magnesium**	Absence of Magnesium
11.	**Ammonium**	Absence of Ammonium
12.	**Mercury**	Absence of Mercury
13.	**Alkaloids**	Absence of Alkaloids
14.	**Reducing Sugar**	Absence of Reducing sugar
15.	**Silicate**	Presence of Silicate
16.	**Copper**	Absence of Copper
17.	**Sodium**	Presence of Sodium
18.	**Lead**	Presence of Lead
19.	**Fluoride And Oxalate**	Presence of Fluoride and Oxalate
20.	**Zinc**	Absence of zinc
21.	**Sulphide**	Presence of sulphide
22.	**Nitrate**	Absence of nitrate
23.	**Nitrite**	Absence of nitrite
24.	**Borate**	Absence of borate
25.	**Arsenic**	Absence of arsenic

QUANTITATIVE ANALYSIS

SOPHISTICATED ANALYTICAL INSTRUMENT FACILITY
IITM,CHENNAI-36
PERKIN ELMER OPTIMA 5300DV ICP-OES

SampleID	Analyte	Mean
Karpoora Silasathu Parpam		
	As193.696	BDL
	Al 308.215	BDL
	B 249.773	250.147 mg/L
	Ca 317.933	458.885 mg/L
	Cd 226.502	BDL
	Cu 324.754	BDL
	Fe 238.204	BDL
	Hg253.652	BDL
	K 766.491	25.124 mg/L
	Mg 257.610	BDL
	Na 588.995	50.249 mg/L
	P 214.914	23.745 mg/L
	Pb 230.204	BDL
	S 181.975	76.235 mg/L
	Si 251.611	19.957 mg/L

BDL=Below detection limit

COLOUR CHARACTERS AND PHYSICOCHEMICAL PROPERTIES OF KARPOORA SILASATHU PARPAM

Table-1

Colour characters of Karpoora Silasathu Parpam

S No	Solvent used	Under ordinary light	Under ultra violet light
1	PM	**Ash**	**Ash**

PM-Powdered material

Table-2

Physicochemical properties of Karpoora Silasathu Parpam

S No.	Parameters	Values obtained (%w/w)	Heavy/ toxic metals	
1	Total ash value	8.24	Lead	BDL
2	Acid insoluble ash	0.92	Cadmium	BDL
3	Water soluble ash	5.7	Mercury	BDL
4	Moisture content	10.11	Arsenic	BDL

Table-3

Colour, nature and percent yields of extracts of Karpoora Silasathu Parpam

S.no.	Extract Solvents	Colour	Nature	% Yield(w/w)	SEM-Micro graph partical size range in micron	pH
1	Water	ash	**Solid**	46	1.5 -3 micron	8.8 -9.1

HR SEM-METHODOLOGY:

An SEM is essentially a high magnification microscope, which uses a focussed scanned electron beam to produce images of the sample, both top-down and, with the necessary sample preparation, cross-sections. The primary electron beam interacts with the sample in a number of key ways:-

- Primary electrons generate low energy secondary electrons, which tend to emphasize the topographic nature of the specimen.
- Primary electrons can be backscattered which produces images with a high degree of atomic number (Z) contrast.
- Ionized atoms can relax by electron shell-to-shell transitions, which lead to either X-ray emission or Auger electron ejection. The X-rays emitted are characteristic of the elements in the top few μm of the sample.

SAMPLE PREPARATION:

Sample preparation can be minimal or elaborate for SEM analysis, depending on the nature of the samples and the data required. Minimal preparation includes acquisition of a sample that will fit into the SEM chamber and some accommodation to prevent charge build-up on electrically insulating samples. Most electrically insulating samples are coated with a thin layer of conducting material, commonly carbon, gold, or some other metal or alloy. The choice of material for conductive coatings depends on the data to be acquired: carbon is most desirable if elemental analysis is a priority, while metal coatings are most effective for high resolution electron imaging applications. Alternatively, an electrically insulating sample can be examined without a conductive coating in an instrument capable of "low vacuum" operation.

The SEM is carried out by using FEI-Quanta FEG 200-High Resolution Instrument.

Resolution : 1.2 nm gold particle separation on a carbon substrate

Magnification: From a min of 12x to greater than 1, 00,000 X

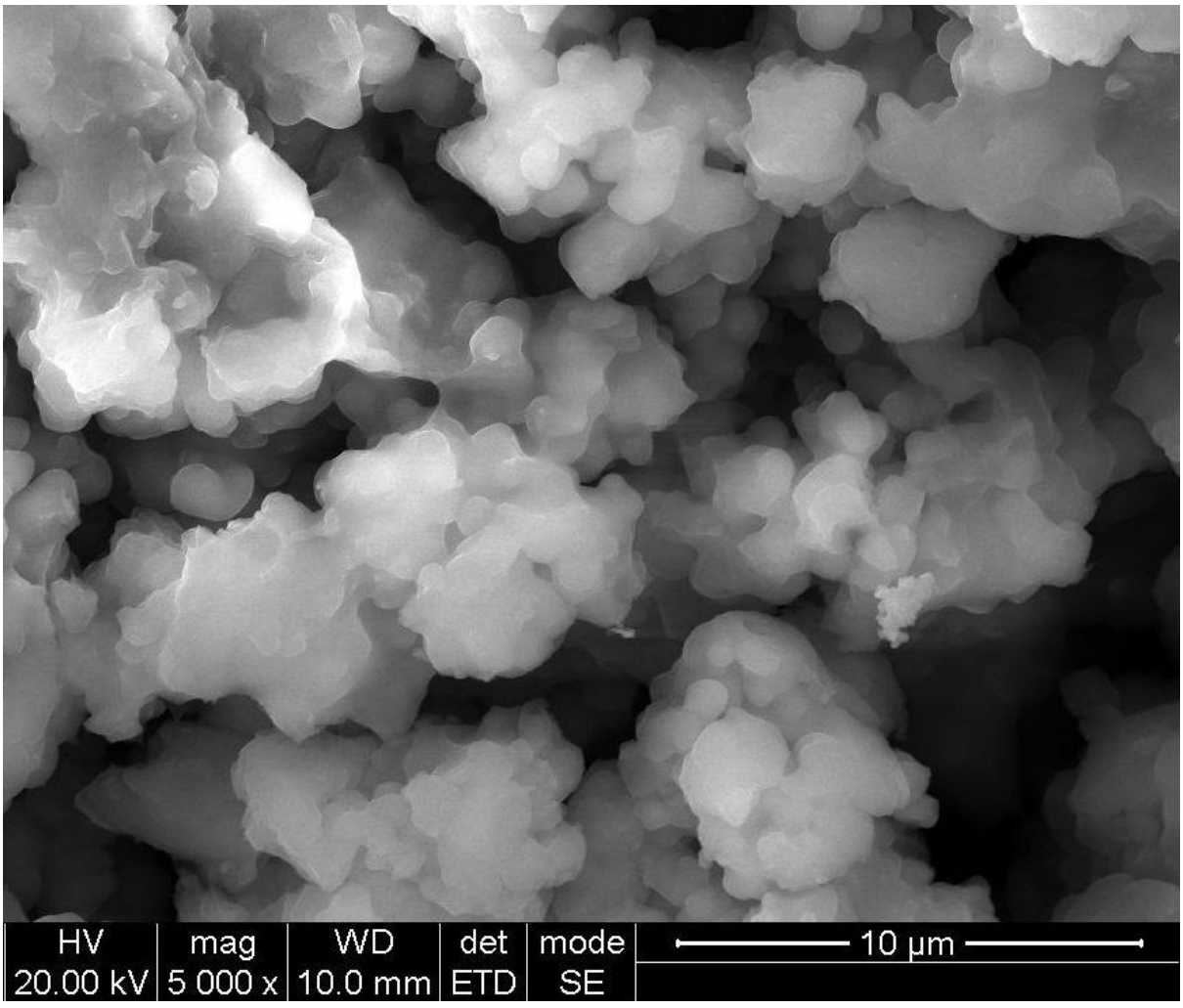
Application : To evaluate grain size, particle size distributions, material homogeneity and inter metallic distributions

.

Experimental Procedure: Done at SAIF, IIT Madras, Chennai-36

SEM – Micro graph partical size

KARPOORA SILASATHU PARPAM



NATIONAL INSTITUTE OF SIDDHA, CHENNAI – 47
AYOTHIDASAR PANDITHAR HOSPITAL
DEPARTMENT OF MARUTHUVAM
PRE CLINICAL AND CLINICAL STUDY ON "AZHAL KALLADAIPPU" (RENAL CALCULI) AND THE DRUG OF CHOICE IS "KARPOORA SILASATHU PARPAM" (INTERNAL)

FORM I - SCREENING AND SELECTION PROFORMA Reg No:32101204/2012-13

1. O.P No _____ 2. I.P No _____ 3. S.No: _____

4. Name: _____ 5. Age (years): 6. Gender: Female/ ☐ Male ☐

7. Contact Nos: ----------

8.INCLUSION CRITERIA:

- Age: 20- 60Yrs of both sex Yes/No
- Clinical symptoms of **abdominal pain & distension, pain from loin to groin, pain in urethra, agonizing pain, dysuria, oliguria, yellow coloured urination, burning micturition, haematuria, nausea & vomiting.** Yes/No
- Stone size**: ≥4mm &≤10mm** Yes/No
- History of Recurrence of Renal calculi Yes/No
- Patient with renal calculus detected on X-ray KUB or USG abdomen. Yes/No
- Patient willing to undergo USG abdomen / X-ray KUB and blood investigations Yes/No
- Patient's willingness for consent to include in the trial Yes/No

9.EXCLUSION CRITERIA:

Stone size >10mm	yes	No	Hepatic disease	Yes	No
Pregnancy and lactation			Other Renal disease		
Severe systemic illness (Eg:CA)			Cardiac disease		
Diabetes mellitus			Hypertension		
Patient taking any other lithotriptic agent			History of taking treatment for any other ailments		
History of drug/alcohol abuse					

10.ADMITTED TO TRAIL: YES ☐ NO ☐ If Yes Serial No:

Date:
Station:
Signature of the Investigator:

Signature of the Lecturer:

Signature of the HOD

NATIONAL INSTITUTE OF SIDDHA, CHENNAI – 47
AYOTHIDASAR PANDITHAR HOSPITAL
DEPARTMENT OF MARUTHUVAM
PRE CLINICAL AND CLINICAL STUDY ON "AZHAL KALLADAIPPU" (RENAL CALCULI) AND THE DRUG OF CHOICE IS "KARPOORA SILASATHU PARPAM" (INTERNAL)

FORM I-A HISTORY PROFORMA ON ENROLLMENT

REG NO: 32101204/2012-13

1. Serial No of the case: _____ 2. OP/IP No: ----------

3. Name: _____ 4. Gender: Female/male

5. Age (years): _____ DOB ☐☐ ☐☐ ☐☐☐☐
Date Month Year

6. Address: ----------

---

7. A) Occupation: ---------- B) Nature of work ----------

8. Educational Status: A) Illiterate ☐ B) Literate ☐

9. Height: --------- cms 10.Weight: ---------- kg

11. Complaints and Duration:

_____

_____

_____

_____

12. Habit of

A) Smoking	1. Yes; duration _____ years; Number-	2.No
B) Tobacco chewing	1. Yes; duration _____ years	2.No
C) Betel chewing	1. Yes; duration _____ years	2.No
D) Alcoholism	1. Yes; duration _____ years; Quantity- ml	2.No

13. Dietary style: A. Pure vegetarian ☐ B. Non-vegetarian ☐ C. mixed diet ☐

14. Drug History: Had the patient been treated before with allopathic drug?
1) Yes ☐ 2) No ☐

15 MARITAL STATUS: 1.Married ☐ 2.Unmarried ☐

No of children: ☐ male: ☐ female: ☐

16. FAMILY HISTORY:

Whether this problem runs in family? 1. Yes ☐ 2.No ☐

If yes, mention the relationship of affected person(s) ----------
---

17. MENSTRUAL HISTORY:

18. BOWEL HABITS & MICTURITION: Normal ☐

History of habitual constipation 1.Yes ☐ 2.No ☐

History of frequent diarrhoea 1.Yes ☐ 2.No ☐

History of frequent dysuria 1.Yes ☐ 2.No ☐

19. PSYCHOLOGICAL STATE:

Normal ☐ Anxiety ☐ Depression ☐

Date:
Station:

Signature of the Investigator:

Signature of the Lecturer:

Signature of the HOD

NATIONAL INSTITUTE OF SIDDHA, CHENNAI – 47
AYOTHIDASAR PANDITHAR HOSPITAL
DEPARTMENT OF MARUTHUVAM
PRE CLINICAL AND CLINICAL STUDY ON "AZHAL KALLADAIPPU" (RENAL CALCULI) AND THE DRUG OF CHOICE IS "KARPOORA SILASATHU PARPAM" (INTERNAL)
FORM II AND II-A CLINICAL ASSESSMENT ON ENROLLMENT AND ON VISITS

1. S. NO ---------- 2. OP/IP NO ---------- REG NO: 32101204/2012-13

3. NAME ---------- 4.GENDER M/F

5. WEEK OF ASSESSMENT :

0^{th} day ☐ 1^{st} week ☐ 2^{nd} week ☐ 3^{rd} week ☐

4^{th} week ☐ 5^{th} week ☐ 6^{th} week ☐ 7^{th} week ☐

SIDDHA SYSTEM OF EXAMINATION:

6. ENVAGAI THERVU:[EIGHT-FOLD EXAMINATION]

I.NAADI: [PULSE PERCEPTION]

	0^{th} day	**1^{st} week**	**2^{nd} week**	**3^{rd} week**	**4^{th} week**	**5^{th} week**	**6^{th} week**	**7^{th} week**
Vali								
Azhal								
Iyyam								
Vali Azhal								
Azhal vali								
Iyya vali								
Vali Iyyam								
Azhal Iyyam								
Iyya Azhal								

II.NAA:[TONGUE]

	0^{th} day	**1^{st} week**	**2^{nd} week**	**3^{rd} week**	**4^{th} week**	**5^{th} week**	**6^{th} week**	**7^{th} week**
Colour	normal/ Red pale/yellow	normal/ Red pale/yellow	normal/ Red pale/yellow	normal/ Red pale/yellow	normal/ Red pale/yellow	normal/ Red pale/yellow	normal/ Red pale/yellow	normal/ Red pale/yellow
Taste	Sweet/Sour/ Pungent/ Bitter/None	Sweet/Sour/ Pungent/ Bitter/None	Sweet/Sour/ Pungent/ Bitter/None	Sweet/Sour/ Pungent/ Bitter/None	Sweet/Sour/ Pungent/ Bitter/None	Sweet/Sour/ Pungent/ Bitter/None	Sweet/Sour/ Pungent/ Bitter/None	Sweet/Sour/ Pungent/ Bitter/None
Coating	Present/ Absent	Present/ Absent	Present/ Absent	Present/ Absent	Present/ Absent	Present/ Absent	Present/ Absent	Present/ Absent
Fissure	Present/ Absent	Present/ Absent	Present/ Absent	Present/ Absent	Present/ Absent	Present/ Absent	Present/ Absent	Present/ Absent
Saliva	Normal/ Increased/ Decreased	Normal/ Increased/ Decreased	Normal/ Increased/ Decreased	Normal/ Increased/ Decreased	Normal/ Increased/ Decreased	Normal/ Increased/ Decreased	Normal/ Increased/ Decreased	Normal/ Increased/ Decreased
Dryness	Present/ Absent	Present/ Absent	Present/ Absent	Present/ Absent	Present/ Absent	Present/ Absent	Present/ Absent	Present/ Absent
Glossitis	Present/ Absent	Present/ Absent	Present/ Absent	Present/ Absent	Present/ Absent	Present/ Absent	Present/ Absent	Present/ Absent
Baldness	Present/ Absent	Present/ Absent	Present/ Absent	Present/ Absent	Present/ Absent	Present/ Absent	Present/ Absent	Present/ Absent

III.NIRAM:[COMPLEXION]

0th day	1st week	2nd week	3rd week	4th week	5th week	6th week	7th week
Dark/pale/ Yellow tinted/ wheatish brown	Dark/pale/ Yellow tinted/ wheatish brown	Dark/pale/ Yellow tinted/ wheatish brown	Dark/pale/ Yellow tinted/ wheatish brown	Dark/pale/ Yellow tinted/ wheatish brown	Dark/pale/ Yellow tinted/ wheatish brown	Dark/pale/ Yellow tinted/ wheatish brown	Dark/pale/ Yellow tinted/ wheatish brown

IV.MOZHI:[VOICE]

0th day	1st week	2nd week	3rd week	4th week	5th week	6th week	7th week
Medium/ High/ Low pitched	Medium/ High/ Low pitched	Medium/ High/ Low pitched	Medium/ High/ Low pitched	Medium/ High/ Low pitched	Medium/ High/ Low pitched	Medium/ High/ Low pitched	Medium/ High/ Low pitched

V.VIZHI:[EYES] (Lower palpebral conjunctiva)

0th day	1st week	2nd week	3rd week	4th week	5th week	6th week	7th week
normal/ Red pale/yellow	normal/Red pale/yellow	normal/Red pale/yellow	normal/ Red pale/yellow	normal/ Red pale/yellow	normal/ Red pale/yellow	normal/Red pale/yellow	normal/Red pale/yellow

VI. MALAM:[BOWEL HABITS / STOOLS]

	0th day	1stweek	2ndwk	3rd week	4th week	5th week	6th week	7th week
Colour	Dark/pale/ yellow/ Red	Dark/pale/ yellow/ Red	Dark/pale/ Yellow/ Red	Dark/pale/ yellow/ Red	Dark/pale/ yellow/ Red	Dark/pale/ yellow/ Red	Dark/pale/ yellow/ Red	Dark/pale/ yellow/ Red
Consistency	Solid/ Semisolid/ Watery	Solid/ Semisolid/ Watery	Solid/ Semisolid/ Watery	Solid/ Semisolid/ Watery	Solid/ Semisolid/ Watery	Solid/ Semisolid/ Watery	Solid/ Semisolid/ Watery	Solid /Semisolid/ Watery
stool bulk	Normal/ Reduced	Normal/ Reduced	Normal/ Reduced	Normal/ Reduced	Normal/ Reduced	Normal/ Reduced	Normal/ Reduced	Normal/ Reduced
Constipation	Present/ Absent	Present/ Absent	Present/ Absent	Present/ Absent	Present/ Absent	Present/ Absent	Present/ Absent	Present/ Absent
Diaarhoea	Present/ Absent	Present/ Absent	Present/ Absent	Present/ Absent	Present/ Absent	Present/ Absent	Present/ Absent	Present/ Absent

VII.MOOTHIRAM:[URINE EXAMINATION]

Neerkkuri	0th day	1st week	2nd week	3rd week	4th week	5th week	6th week	7th week
Niram[Colour]	Yellow/ Red/ White/ Straw coloured/ Crystal clear	Yellow/ Red/ White/ Straw coloured/ Crystal clear	Yellow/ Red/ White/ Straw coloured/ Crystal clear	Yellow/ Red/ White/ Straw coloured/ Crystal clear	Yellow/ Red/ White/ Straw coloured/ Crystal clear	Yellow/ Red/ White/ Straw coloured/ Crystal clear	Yellow/ Red/ White/ Straw coloured/ Crystal clear	Yellow/ Red/ White/ Straw coloured/ Crystal clear
Manam[Odour]	Present/ Absent	Present/ Absent	Present/ Absent	Present/ Absent	Present/ Absent	Present/ Absent	Present/ Absent	Present/ Absent
Nurai[Froth]	Nil/ Reduced/ Increased	Nil/ Reduced/ Increased	Nil/ Reduced/ Increased	Nil/ Reduced/ Increased	Nil/ Reduced/ Increased	Nil/ Reduced/ Increased	Nil/ Reduced/ Increased	Nil/ Reduced/ Increased
Edai[Sp.gravity]	Normal/ Increased/ Reduced	Normal/ Increased/ Reduced	Normal/ Increased/ Reduced	Normal/ Increased/ Reduced	Normal/ Increased/ Reduced	Normal/ Increased/ Reduced	Normal/ Increased/ Reduced	Normal/ Increased/ Reduced
Enjal[Deposits]	Present/ Absent	Present/ Absent	Present/ Absent	Present/ Absent	Present/ Absent	Present/ Absent	Present/ Absent	Present/ Absent
Volume	Normal/ Increased/ Reduced	Normal/ Increased/ Reduced	Normal/ Increased/ Reduced	Normal/ Increased/ Reduced	Normal/ Increased/ Reduced	Normal/ Increased/ Reduced	Normal/ Increased/ Reduced	Normal/ Increased/ Reduced

Neikkuri	0th day	1st week	2nd week	3rd week	4th week	5th week	6th week	7th week
Serpentine fashion	at___mints	at___ mints	at___ mints	at___ mints	at___ mints	at__mints	at__mints	at___ mints
Annular/Ringed fashion	at___mints	at___ mints	at___ mints	at___ mints	at___ mints	at__mints	at__mints	at___ mints
Pearl beaded fashion	at___mints	at___ mints	at___ mints	at___ mints	at___ mints	at__mints	at__mints	at___ mints
Mixed fashion	at___mints	at___ mints	at___ mints	at___ mints	at___ mints	at__mints	at__mints	at___ mints
Other fashion	at___mints	at___ mints	at___ mints	at___ mints	at___ mints	at__mints	at__mints	at___ mints

VIII. SPARISAM:[PALPATORY PERCEPTION]

0th day	1st week	2nd week	3rd week	4th week	5th week	6th week	7th week
Warmth/Hot/ cold/ Sweat	Warmth/Hot /cold/ Sweat	Warmth/Hot /cold/ Sweat	Warmth/Hot/ cold/ Sweat	Warmth/Hot/ cold/ Sweat	Warmth/Hot/ cold/ Sweat	Warmth/Hot/ cold/ Sweat	Warmth/Hot/ cold/ Sweat

7. THEGI:[TYPE OF BODY CONSTITUTION]

Vatham predominant		Kabam predominant	
Pitham predominant		Thondha udal	

8. NILAM:[LAND WHERE PATIENT LIVED MOST]

Kurinji ☐ Mullai ☐ Marutham ☐ Neithal ☐ Palai ☐

(Hilly terrain) (Forest range) (Plains) (Coastal belt) (Arid regions)

9. KAALAM: [SEASON]

Kaarkalam ☐ Pinpanikalam ☐

Koothirkalam ☐ Ilavenil ☐

Munpanikalam ☐ Muthuvenil ☐

10. GUNAM:[CHARACTER]

Sathuvam ☐ Rasatham ☐ Thamasam ☐

11. IYMPORIGAL:[SENSORY ORGANS]

	0th day	1st week	2nd week	3rd week	4th week	5th week	6th week	7th week
	Normal/ Affected	Normal/ Affected	Normal/ Affected	Normal/ Affected	Normal/ Affected	Normal/ Affected	Normal/ Affected	Normal/ Affected
Mei [Skin]								
Vaai[Buccal cavity]								
Kan [Eyes]								
Mooku[Nose]								
Sevi [ear]								

12. IYMPULANGAL: [MOTOR ORGANS]

	0th day	1st week	2nd week	3rd week	4th week	5th week	6th week	7th week
	Normal/ Affected	Normal/ Affected	Normal/ Affected	Normal/ Affected	Normal/ Affected	Normal/ Affected	Normal/ Affected	Normal/ Affected
Kai [upperlimb]								
Kal [lowerlimb]								
Vai[Buccal cavity]								
Eruvai [excretory organ]								
Karuvai[Reproduc-tive organ]								

13. KOSAM: [SHEATHS]

	0th day	1st week	2nd week	3rd week	4th week	5th week	6th week	7th week
	Normal/ Affected	Normal/ Affected	Normal/ Affected	Normal/ Affected	Normal/ Affected	Normal/ Affected	Normal/ Affected	Normal/ Affected
Annamaya kosam								
Pranamaya kosam								
Manonmayakosam								
Vingyanamaya kosam								
Anandhamaya kosam								

14. MUKKUTRAM:[AFFECTION OF THREE HUMORS]

A) VATHAM:

	0th day	1st week	2nd week	3rd week	4th week	5th week	6th week	7th week
	Normal/ Affected	Normal/ Affected	Normal/ Affected	Normal/ Affected	Normal/ Affected	Normal/ Affected	Normal/ Affected	Normal/ Affected
Praanan								
Abaanan								
Samaanan								
Udhaanan								
Viyaanan								
Naahan								
Koorman								
Kirukaran								
Devathathan								
Dhananjeyan								

B) PITHAM:

	0th day	1st week	2nd week	3rd week	4th week	5th week	6th week	7th week
	Normal/ Affected	Normal/ Affected	Normal/ Affected	Normal/ Affected	Normal/ Affected	Normal/ Affected	Normal/ Affected	Normal/ Affected
Analapitham								
Prasakam								
Ranjakam								
Aalosakam								
Saathakam								

C) KABAM:

	0th day	1st week	2nd week	3rd week	4th week	5th week	6th week	7th week
	Normal/ Affected	Normal/ Affected	Normal/ Affected	Normal/ Affected	Normal/ Affected	Normal/ Affected	Normal/ Affected	Normal/ Affected
Avalambagam								
Kilethagam								
Pothagam								
Tharpagam								
Santhigam								

15. SEVEN DHATHUS: [SEVEN SOMATIC COMPONENTS]

	0th day	1st week	2nd week	3rd week	4th week	5th week	6th week	7th week
	Normal/ Affected	Normal/ Affected	Normal/ Affected	Normal/ Affected	Normal/ Affected	Normal/ Affected	Normal/ Affected	Normal/ Affected
Saaram[chyme]								
Senneer[Blood]								
Oon[Muscle]								
Kozhuppu[Fat]								
Enbu[Bones]								
Moolai[Bonemarrow]								
Sukkilam/Suronitham [Genital discharges]								

16. SYSTEMIC EXAMINATION:

	0th day	1st week	2nd week	3rd week	4th week	5th week	6th week	7th week
CardioVascular System								
Respiratory System								
Gastrointestinal System								
CentralNervous System								
Endocrine System								

UROGENITAL SYSTEM	0th day	1st week	2nd week	3rd week	4th week	5th week	6th week	7th week
Nephrotic syndrome								
Glomerulo Nephritis								
Acute/chronic Interstitial Nephritis								
Acute/chronic Renal failure								
Polycystic kidneydisease								

17. GENERAL EXAMINATION:

	0th day	1st week	2nd week	3rd week	4th week	5th week	6th week	7th week
Height (cms)								
Weight (kg)								
Temperature(°F)								
Pulse rate (per min)								
Heart rate (per min)								
Respiratory rate(per min)								
Blood pressure(mm/Hg)								
Pallor								
Jaundice								
Cyanosis								
Lymphadenopathy								
Pedal edema								
Clubbing								
Jugular vein pulsation								

18. CLINICAL SYMPTOMS:

	0th day	1st week	2nd week	3rd week	4th week	5th week	6th week	7th week
Abdominal pain								
Pain from loin to groin								
Agonizing pain								
Pain in urethra								
Yellow coloured urination								
Burning micturition								
Oliguria								
Dysuria								
Abdominal distention								
Nausea & Vomiting								
Haematuria								

Date:
Station:
Signature of the Investigator:

Signature of the Lecturer:

Signature of the HOD

NATIONAL INSTITUTE OF SIDDHA, CHENNAI – 47
AYOTHIDASAR PANDITHAR HOSPITAL
DEPARTMENT OF MARUTHUVAM

PRE CLINICAL AND CLINICAL STUDY ON "AZHAL KALLADAIPPU" (RENAL CALCULI) AND THE DRUG OF CHOICE IS "KARPOORA SILASATHU PARPAM" (INTERNAL)

FORM III LABORATORY PARAMETERS-CHART

1. OP/IP No: _____ 2.S. No: _____ 3.Reg no: 32101204/2012-13
4. Name: _____ 5. Age: _____ years 6. Gender: M/F

BLOOD INVESTIGATION		0th DAY Date:	49th DAY Date:	NORMAL VALUES
HB (gms%)				M:14-18 ;W:11-15
T.RBC(milli/cu.mm)				M:4.5-6.5 ;W:3.5-5.5
ESR (mm)	½ hr.			
	1 hr.			M:0-10 ;W:0-20
T.WBC (cu.mm)				4000-11,000
DIFFERENTIAL COUNT (%)	Polymorphs			40-75
	Lymphocytes			20-35
	Monocytes			2-10
	Eosinophils			1-6
	Basophils			0-1

BT (per min)				2-6
Clotting time				3-8
Blood glucose (mg/dl)	Fasting			80-120
	PP			<130
	Random			<140
Lipid profile (mg/dl)	Serum cholesterol			150-250
	HDL			30-60
	LDL			Upto 130
	VLDL			40
	TGL			Upto 160
RFT (mg/dl)	Blood urea			16-50
	Serum creatinine			0.6-1.2
	Serum Uric acid			M:3-9 ;W: 2.5-7.5
LFT (mg/dl)	Total bilirubin			0.3-1
	Direct bilirubin			0.1-0.3
	Indirect bilirubin			0.2-0.8
	Serum total protein			6-8
	Serum Albumin			3.5-5.5
	Serum globulin			2-3.5
	Fibrinogen(g/dl)			0.2-0.4
	Serum calcium			9-11
	Serum phosphorous			2-5
	SGOT (IU/L)			6-18
	SGPT (IU/L)			3-26
	Alkaline phosphatase (kingÅ units)			3-12

URINE INVESTIGATION	Before TMT Date:	After TMT Date:
Neerkkuri		
Niram		
Manam		
Nurai		
Edai		
Enjal		
Neikkuri		
Albumin		
Fasting sugar		
PP sugar		
Random Sugar		
Deposits		
Bile salts		
Bile pigments		
Urobilinogen		
Culture & sensitivity		
MALAM		
Ova		
Cyst		
Occult blood		

SCAN: USG ABDOMEN

Specific investigation		Size of the kidney	site of the calculus	No of calculus	Size of the calculus	Hydro nephrosis
Rt kidney	Before treatment (0^{th} day)					
	After treatment (49^{th} day)					
Lt kidney	Before treatment (0^{th} day)					
	After treatment (49^{th} day)					

X RAY CHANGES:

	Before treatment (0^{th} day)	After treatment (49^{th} day)
X RAY – KUB		

Date:

Station:

Signature of the Investigator:

Signature of the Lecturer:

Signature of the HOD

NATIONAL INSTITUTE OF SIDDHA, CHENNAI – 47
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DEPARTMENT OF MARUTHUVAM
PRE CLINICAL AND CLINICAL STUDY ON "AZHAL KALLADAIPPU" (RENAL CALCULI) AND THE DRUG OF CHOICE IS "KARPOORA SILASATHU PARPAM" (INTERNAL)
FORM IV A (DRUG COMPLIANCE FORM)

S. NO: --------- OPD/IPD NO: ---------- NAME :---------- REG NO: 32101204/2012-13

Name Of The Drug: karpoora silasathu parpam, 130mg, twice a day with Radish juice

On 1st week -Date: ; Drugs issued: (Nos) / Drugs returned: (Nos)
On 2nd week-Date: ; Drugs issued: (Nos) / Drugs returned: (Nos)
On 3rd week -Date: ; Drugs issued: (Nos) / Drugs returned: (Nos)
On 4th week-Date: ; Drugs issued: (Nos) / Drugs returned: (Nos)
On 5th week -Date: ; Drugs issued: (Nos) / Drugs returned: (Nos)
On 6th week-Date: ; Drugs issued: (Nos) / Drugs returned: (Nos)
On 7th week -Date: ; Drugs issued: (Nos) / Drugs returned: (Nos)

Day	Date/ தேதி	Morning/ காலை	Evening/ மாலை	Day	Date/தேதி	Morning/ காலை	Evening/ மாலை
Day 1				Day25			
Day2				Day26			
Day3				Day27			
Day4				Day28			
Day5				Day29			
Day6				Day30			
Day7				Day31			
Day8				Day32			
Day9				Day33			
Day10				Day34			
Day11				Day35			
Day12				Day36			
Day13				Day37			
Day14				Day38			
Day15				Day39			
Day16				Day40			
Day17				Day41			
Day18				Day42			
Day19				Day43			
Day20				Day44			
Day21				Day45			
Day22				Day46			
Day23				Day47			
Day24				Day 48			

Date: Station:

Signature of the Investigator:

Signature of the Lecturer:

Signature of the HOD

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PRE CLINICAL AND CLINICAL STUDY ON "AZHAL KALLADAIPPU" (RENAL CALCULI) AND THE DRUG OF CHOICE IS "KARPOORA SILASATHU PARPAM" (INTERNAL)

FORM IV-B INFORMATION SHEET

Name of the Principal Investigator: Dr.S.Raja Rajeswari

Name of the Institution : National Institute of Siddha
Tambaram Sanatorium
Chennai- 47.

- I, Dr.S.Raja Rajeswari studying M.D (Siddha) in National Institute of Siddha, Chennai. The disease called **Azhal kalladaippu (Renal calculi)** is formed by the sedimentation of crystalline mineral materials within the kidney or urinary tract. It includes the symptoms like, pain in the back region, radiating towards the groins and lower abdomen, painful urination, scanty urination, nausea, vomiting and may also pass the urine with blood. This condition is being treated in NIS with many siddha formulations. As a part of M.D(S) research programme and developing new efficacious medicine, we propose to study the **karpoora silasathu parpam** formulation for treating the condition. This formulation has been mentioned in siddha literature and empirical evidence with contemporary tools is required for documentation. You can receive medicines free of cost. The duration of treatment period is 48 days. You have to visit NIS every week and collect drugs for **7 days.** The diagnosis tests will be carried out free of cost. However, a particular test **USG Abdomen** has to be taken from outside lab and charges to be borne by you. We will assess the effect of treatment after completion of **48 days** of treatment using clinical and lab parameters.
- In this regard, we need to ask you few questions. We will maintain confidentiality of your comments and data obtained from you. There will be no risk of disclosing your identity and no physical, psychological or professional risk is involved by taking part in this study.
- Taking part in this study is voluntary. No compensation will be paid to you for taking part in this study. You can choose not to answer any specific question. There is no specific benefit for you if you take part in the study, but you will be under our clinical monitoring and specific attention will be given for your health. Taking part in the study may be of benefit to the community, as it may help us to develop medicine for Azhal kalladaippu. In case of any adverse symptoms during the treatment viz, acute renal colic i.e. severe pain caused by the kidney stone and associated with nausea, vomiting and fever which are expected in few patients during the treatment, shall be reported to PIs and care will be taken in NIS for relief. You can withdraw from the study at the midst of treatment period, if you are not interested to continue and you will receive our usual treatment without condition.
- The information we will collect in this study, will remain between you and the principal investigator. We will ask you a few questions through questionnaire. We will not write your name on different forms which sent to different investigating/analysis sections and we will use a code instead given by the principal investigator. Only the principal investigator will know the key to this code which will be kept in safe custody. If you agree to be a participant in this study, you will be screened as per the study protocol.
- If you wish to find out more about this study before taking part, you can ask me all the questions you want or contact Dr.S.Raja Rajeswari, PG scholar cum principal investigator of this study, attached to the National Institute of Siddha, Chennai (Mobile phone no:9486770094). You can also contact the Chairman/Member-secretary of Ethics committee, National Institute of Siddha, Chennai – 600047, Tel no: 91-44-22411611, for rights and participation in the study.

தேசிய சித்த மருத்துவ நிறுவனம், சென்னை 47
அயோத்திதாசர் பண்டிதர் மருத்துவமனை
அழல் கல்லடைப்பு நோய்க்கான சித்த மருந்தின் (கற்ப்பூரசிலாசத்து பற்பம்) பரிகரிப்புத் திறனைக் கண்டறியும் மருத்துவ ஆய்விற்கான தகவல் படிவம்.

FORM IV-B தகவல் படிவம்

முதன்மை ஆராய்ச்சியாளர் பெயர் : Dr. செள.ராஜராஜேஸ்வரி
நிறுவனத்தின் பெயர் : தேசிய சித்த மருத்துவ நிறுவனம்
தாம்பரம் சானட்டோரியம்
சென்னை 47

Dr.செள.ராஜராஜேஸ்வரி ஆகிய நான் தேசிய சித்த மருத்துவமனையில் பட்ட மேற்படிப்பு பயின்று வருகிறேன். **கல்லடைப்பு** என்னும் நோயானது சிறுநீரகம் மற்றும் சிறுநீரகப்பாதையில் உப்புக்கள் படிந்து முட்கள்போல் மாறி சிறுநீர் பாதையை அடைத்து வலியையும் சிறுநீர் தடையையும் உண்டு பண்ணும் ஒரு நோயாகும். இந்நோய் இடுப்பினைச் சுற்றிக் குத்தல், வலி, சிறுநீர் செல்லும்போது வலி, எரிச்சல், சிலசமயங்களில் சிறுநீருடன் இரத்தம் கலந்து வெளியாதல் போன்ற குறிகுணங்களைத் தோற்றுவிக்கும். இந்நோய்க்கு தேசிய சித்த மருத்துவமனையில் பல சித்த மருந்துகள் பயன்படுத்தப்பட்டு வருகின்றது. சித்த மருத்துவ பட்ட மேற்படிப்பில், ஆய்வின் ஒரு பகுதியாக புதிய மருந்துகளை பயன்படுத்தும் நோக்கில் **கற்ப்பூர சிலாசத்து பற்பம்** மருந்தினை இந்நோய்க்கு வழங்க பரிந்துரை செய்கிறோம். இந்த மருந்தின் செய்முறை, அளவு, அனுபானம் மற்றும் மருத்துவ பயன்கள் அனைத்தும் அங்கீகரிக்கப்பட்ட சித்த மருத்துவ நூலில் கூறப்பட்டுள்ளது. எந்தவித கட்டணமுமின்றி தாங்கள் இந்த மருந்தினை பெற்றுக்கொள்ளலாம். இந்த ஆய்வில் மருந்து உட்கொள்ளும் காலம் **48நாட்கள்** ஆகும். வாரம் ஒருமுறை தேசிய சித்த மருத்துவமனைக்கு நேரில் வந்து 7நாட்களுக்கான மருந்தினை பெற்றுக்கொள்ள வேண்டும். இந்த ஆய்வு சம்பந்தமான ஆய்வக பரிசோதனைகள் கட்டணமின்றி செய்யப்படும். மேலும் இந்நோய்க்கான, குறிப்பிட்ட **USG ABDOMEN** பரிசோதனை வெளி ஆய்வுக்கூடத்தில் தங்கள் செலவிலேயே செய்து கொள்ள வேண்டும். 48நாட்கள் மருந்து உட்கொள்ளும் காலம் முடிந்த பிறகு நோய்க்கான குறிகுணங்கள் மற்றும் ஆய்வக பரிசோதனைகள் இவற்றின் முடிவுகளின் அடிப்படையில் மருந்தின் பரிகரிப்புத்திறன் கண்டறியப்படும்.

இந்த ஆய்வு சம்பந்தமாக சில கேள்விகளை தங்களிடம் கேட்க இருக்கிறேன். தங்களிடமிருந்து பெறப்படும் கருத்துக்கள் மற்றும் குறிப்புகள் அனைத்தும் நம்பிக்கையாக பதிவு செய்யப்படும்.இந்த ஆய்வில் தங்களை உட்படுத்திக்கொள்வதின் மூலம் எந்த வகையிலும் பாதிப்புக்குள்ளாக மாட்டீர்கள் என உறுதி அளிக்கிறேன்.

எந்தவித வற்புறுத்தலுமின்றி, இந்த ஆய்வில் பங்கேற்கவும், இந்த ஆய்வு சம்பந்தமாக கேட்கப்படும் கேள்விகளுக்கு பதில் கூறவும் தங்களுக்கு முழு சுதந்திரம் அளிக்கப்படுகிறது. இந்த ஆய்வில் பங்கேற்பதற்கு எந்த சன்மானமும் வழங்கப்படமாட்டாது. ஆனால், ஆய்வு முழுவதும் எனது மேற்பார்வையிலும், தங்கள் உடல்நலன் குறித்த தனி கவனத்திலும் ஆய்வு மேற்கொள்ளப்படும்.கல்லடைப்பு நோய்க்கான புதிய மருந்தின் பரிகரிப்புத்திறனை சமூகத்திற்கு உணர்த்தும் வகையில் இந்த ஆய்வு மேற்கொள்ளப்படுகிறது. இந்த ஆய்வில், மருந்து உட்கொள்ளும் காலத்தில் சிலருக்கு இடுப்பைச்சுற்றிலும் தாங்கமுடியாத வலியுடன் குமட்டல், வாந்தி, சுரம் போன்ற மாறுபட்ட குறிகுணங்கள் தொடர்ந்து இருக்கும் பட்சத்தில், முதன்மை ஆராய்ச்சியாளரான என்னிடம் தெரிவிக்கப்பட்டு, தேசிய சித்த மருத்துவமனையில் அதற்க்கான தீர்வு வழங்கப்படும். இந்த ஆய்வினைத் தொடர தங்களுக்கு விருப்பம் இல்லையெனில், எப்பொழுது வேண்டுமானலும் ஆய்வின் இடையில் விலகிக்கொள்ளவும், இம்மருத்துவமனையில் வழங்கப்படும் இந்நோய்க்கான வழக்கமான மருந்துகளை பெற்றுக்கொள்ளவும் அறிவுறுத்தப்படுகிறீர்கள்.

இந்த ஆய்வில் சேகரிக்கப்படும் விபரங்கள் அனைத்தும் தங்களுக்கும் முதன்மை ஆராய்ச்சியாளரான எனக்கும் இடையில் இரகசியமாக வைக்கப்படும். கேள்வி பதில் வடிவத்தில் தங்களிடம் கேள்விகள் கேட்கப்படும். அனைத்துப் படிவங்களிலும் தங்களின் பெயர் தவிர்க்கப்பட்டு ஆய்வாளரால் தங்களுக்கென தனிக் குறியீடு வழங்கப்படும். அந்தக் குறியீடு ஆய்வாளருக்கு மட்டுமே தெரிந்ததாக இருக்கும். நீங்கள் இந்த ஆய்வில் பங்கேற்க விருப்பப்பட்டால், திட்ட வரைவு படி தேர்வு செய்யப்படுவீர்கள்.

நீங்கள் இந்த ஆய்வில் பங்கேற்கும் முன், இந்த ஆய்வினைப் பற்றிய மேலும் விபரங்கள் பெற வேண்டுமென விருப்பப்பட்டால், இந்த ஆய்வின் முதன்மை ஆராய்ச்சியாளர் மற்றும் தேசிய சித்த மருத்துவமனை, பட்ட மேற்படிப்புத்துறை மாணவர் Dr.செள.ராஜராஜேஸ்வரி ஆகிய என்னை 9486770094 என்ற எண்ணில் தொடர்பு கொள்ளலாம். மேலும், நீங்கள் இந்த ஆய்வில், உங்களது பங்கேற்பு மற்றும் உரிமை பற்றி தெரிந்து கொள்ள தேசிய சித்த மருத்துவமனை, தலைவர்/செயற்க்குழு உறுப்பினர் அவர்களையும் 91-44-22411611 என்ற எண்ணில் தொடர்பு கொள்ளலாம்.

NATIONAL INSTITUTE OF SIDDHA, CHENNAI – 47
AYOTHIDASAR PANDITHAR HOSPITAL
DEPARTMENT OF MARUTHUVAM

PRE CLINICAL AND CLINICAL STUDY ON "AZHAL KALLADAIPPU" (RENAL CALCULI) AND THE DRUG OF CHOICE IS "KARPOORA SILASATHU PARPAM" (INTERNAL)

FORM IV-C CONSENT FORM

CERTIFICATE OF CONSENT

"I have read the foregoing information, or it has been read to me. I have had the opportunity to ask questions about it and any questions I have asked have been answered to my satisfaction.

I consent voluntarily to participate as a participant in this study and understand that I have the right to withdraw from the study at any time without in any way it affecting my further medical care".

"I have received a copy of the information sheet/consent form".

In case of illiterate participant

"I have witnessed the accurate reading of the consent form to the potential participant, and the individual has had the opportunity to ask questions. I confirm that the individual has given consent freely."

Date:

Signature of a witness

Left thumb Impression of the Participant

(Selected by the participant bearing no connection with the survey team)

Date:
Station:
Signature of participant:

Signature of the Investigator:

Signature of the Lecturer:

Signature of the HOD

தேசிய சித்த மருத்துவ நிறுவனம்
அயோத்திதாச பண்டிதர் மருத்துவமனை, சென்னை - 47.
பட்ட மேற்படிப்பு மருத்துவத்துறை

அழல் கல்லடைப்பு நோய்க்கான சித்த மருந்தின் (கற்ப்பூர சிலாசத்து பற்பம்) பரிகரிப்புத் திறனைக் கண்டறியும் மருத்துவ ஆய்விற்கான ஒப்புதல் படிவம்

FORM IV- C ஒப்புதல் படிவம்

நான் மேற்கூறிய தகவல் படிவத்தை படித்து அல்லது படிக்க கேட்டு கொண்டேன். து தொடர்பான விளக்கங்களையும் கேட்டு தெரிந்து கொண்டேன். எந்த வித வற்புறுத்தலின்றி, என் சொந்த விருப்பத்தின் பேரில் என்னை ந்த ஆராய்ச்சிக்கு உட்படுத்த என் முழுமனதோடும் சுயநினைவோடும் சம்மதம் தெரிவிக்கிறேன். எனக்கு விருப்பமில்லாத பட்சத்தில் இந்த ஆராய்ச்சியில் இருந்து என்னை எப்போதுவேண்டுமானாலும் விடுவித்து கொள்ளும் உரிமையை பெற்றுள்ளேன் என்பதையும் அறிவேன்.

தேதி: கையொப்பம்:

இடம்: பெயர் :

தேதி: சாட்சிக்காரர் கையொப்பம்:
இடம்: பெயர் :
உறவுமுறை :

NATIONAL INSTITUTE OF SIDDHA, CHENNAI – 47

AYOTHIDASAR PANDITHAR HOSPITAL

DEPARTMENT OF MARUTHUVAM

PRE CLINICAL AND CLINICAL STUDY ON "AZHAL KALLADAIPPU" (RENAL CALCULI) AND THE DRUG OF CHOICE IS "KARPOORA SILASATHU PARPAM" (INTERNAL)

FORM IV- D DIETARY ADVICE FORM

The following diet to be taken:

- For a day, drink 2- 3 liters of water.
- Should take Barley rice kanji, kol kudineer and tender coconut.

- **Vegetables:**

  Radish, Indian butter bean, Lady's finger, Bitter bottle gourd, White pumpkin, Green's stem, Plantain stem, Ribbed gourd, Carrot.

- **Greens:**

  Brown Indian hemp, Marsh mint, Curry leaf.

- **Fruits:**

  Watermelon, Cucumber, Pineapple, Lemon, Papaya, Banana.

- **Grains:**

  Bengal gram, black gram, green gram, pea.

The following food should be avoided:

- Tomato
- Mushrooms
- Cauliflower
- Chocolate
- Greens
- Licker
- Strawberry
- Tamarind
- betel-nut
- Coffee/Tea
- Cabbage
- fish
- drumstick
- Meat
- Egg
- Grapes
- baking soda
- Betel leaf
- Tobacco

- preserved cool drinks
- excessive intake of salt content
- milk products
- fried and spicy foods

தேசிய சித்த மருத்துவ நிறுவனம்
அயோத்திதாஸர் பண்டிதர் மருத்துவமனை,சென்னை-47
பட்டமேற்படிப்பு மருத்துவத்துறை
அழல் கல்லடைப்பு நோய்க்கான சித்த மருந்தின் (கற்பூர சிலாசத்து பற்பம்) பரிகரிப்புத் திறனைக் கண்டறியும் மருத்துவ ஆய்விற்கான உணவு அறிவுரை படிவம்

FORM IV- D உணவு அறிவுரை படிவம்

சேர்க்கவேண்டியவை:

- நாள் ஒன்றுக்கு **2 முதல் 3 லிட்டர்** வரை தண்ணீர் அருந்த வேண்டும்.
- பார்லி அரிசிக் கஞ்சி, கொள்ளு அவித்த நீர், இளநீர் ஆகியவை சேர்க்க வேண்டும்.

காய்கள்:

முள்ளங்கி, அவரை, வெண்டை, சுரைக்காய், வெண்பூசணி, கீரைத்தண்டு, வாழைத்தண்டு, கேரட், பீர்க்கங்காய், ஆகியவை சேர்க்க வேண்டும்.

கீரைகள்:

சிறுகீரை, தாளிக்கீரை, காசினிக்கீரை, புதினாக்கீரை, கருவேப்பிலை ஆகியவை சேர்க்க வேண்டும்.

பழங்கள்:

தர்பூசணி, வெள்ளரிப்பிஞ்சு, அன்னாசிப்பழம், பப்பாளி, வாழைப்பழம், எலுமிச்சை ஆகியவை சேர்க்க வேண்டும்.

தானியங்கள்:

கடலைப்பருப்பு. உளுந்து, பாசிப்பயறு, உலர்ந்த பட்டாணி ஆகியவை சேர்க்க வேண்டும்.

தவிர்க்க வேண்டியவை:

➢ தக்காளி	முட்டைக்கோசு
➢ காளிபிளவர்	காளான்கள்
➢ காபி/டீ	மீன்
➢ சாக்லேட்	மாமிச உணவு
➢ கீரைகள்	முருங்கைக்காய்
➢ முட்டை	மதுபானம்
➢ திராட்சை	ஸ்ட்ராபெர்ரி
➢ சமையல்சோடா	புளி
➢ வெற்றிலை/பாக்கு	புகையிலை

- ➢ பதப்படுத்தப்பட்ட குளிர் பானங்கள்
- ➢ உப்பு நிறைந்த உணவு மற்றும் நீர்
- ➢ பாலில் தயாரிக்கப்பட்ட உணவு வகைகள்
- ➢ பொரிக்கப்பட்ட மற்றும் மசாலா சேர்ந்த உணவு வகைகள்.

வாழ்க்கை முறை மாற்றங்கள்:

- ➢ ஒரே இடத்தில் வெகுநேரம் நின்று அல்லது அமர்ந்து செய்யும் வேலைகளைத் தவிர்க்கவும். முடியாத பட்சத்தில் ஒரு மணி நேரத்திற்கு ஒருமுறையேனும் சற்றே எழுந்து நடப்பது அவசியம்.
- ➢ சிறுநீரை வெகுநேரம் அடக்குதல் கூடாது. சிறுநீர் கழிக்க வேண்டும் என்ற உந்துதல் ஏற்பட்ட உடனே அதை கழித்து விட வேண்டும்.
- ➢ வெயிலில் சுற்றுவதைக் குறையுங்கள். புழுக்கமான இடங்களில் அதிக நேரம் செலவிட வேண்டாம்.
- ➢ நீண்ட தூர பயணங்களைத் தவிர்ப்பது நல்லது.
- ➢ ஓய்வும் தூக்கமும் மிக மிக அவசியம்.
- ➢ மன அழுத்தம், மன இறுக்கம் போன்றவை இருந்தால் யோகா, தியானம் போன்ற முறைகளைக்
- ➢ கற்றுக்கொண்டு அதைக் குறைக்கவும்.
- ➢ பரபரப்பான, ஓய்வில்லாத வாழ்க்கை முறை இருந்தால் அதை உடனடியாக மாற்றி அமைக்கவும்.

NATIONAL INSTITUTE OF SIDDHA, CHENNAI – 47
AYOTHIDASAR PANDITHAR HOSPITAL
DEPARTMENT OF MARUTHUVAM

PRE CLINICAL AND CLINICAL STUDY ON "AZHAL KALLADAIPPU" (RENAL CALCULI) AND THE DRUG OF CHOICE IS "KARPOORA SILASATHU PARPAM" (INTERNAL)

FORM IV -E (WITHDRAWAL FORM)

1)S. NO: ---------- 2) OPD/ IPD NO: ---------- 3)REG NO: 32101204/2012-13

4)NAME:---------- 5) AGE: --------- 6) GENDER: M/F--------

Date of trial commencement:

Date of withdrawal from trial:

REASONS FOR WITHDRAWAL:

- Long absence at reporting : Yes/ No
- Irregular treatment: Yes/ No
- Shift of locality : Yes/No
- Increase in severity of symptoms: Yes/No
- Complication/Adverse reactions if any: Yes/No
- Poor patient compliance: Yes/No

Date:

Station:

Signature of the Investigator:

Signature of the Lecturer:

Signature of the HOD

NATIONAL INSTITUTE OF SIDDHA, CHENNAI – 47
AYOTHIDASAR PANDITHAR HOSPITAL
DEPARTMENT OF MARUTHUVAM

PRE CLINICAL AND CLINICAL STUDY ON "AZHAL KALLADAIPPU" (RENAL CALCULI) AND THE DRUG OF CHOICE IS "KARPOORA SILASATHU PARPAM" (INTERNAL)

FORM IV F

ADVERSE REACTION FORM

Name:

Age:

Gender: Male/Female

Serial No: Op/Ip No:

Reg No: 32101204/2012-13

Date of trial commencement:

Date of the adverse reaction occur:

Time:

Description of Adverse reaction:

Date:

Station:

Signature of the Investigator:

Signature of the Lecturer:

Signature of the HOD

NATIONAL INSTITUTE OF SIDDHA
(An Autonomous Body under Department of AYUSH)
Ministry Of Health & Family Welfare, Government of India

Tambaram Sanatorium, Chennai - 600 047
Tel : 044-22411611 Fax : 044-22381314
E-mail : nischennaisiddha@yahoo.co.in
Website : www.nischennai.org

Name: Dr. S. RAJA RAJESWARI, REG.NO: 32101204
Title: Preclinical and clinical study on Azhal kalladaippu (Renal calculi) and the Drug of choice is 'Karpoora Silasaram Parpam'.
No. NIS/IEC/2011/3/04 - 24/12/2011

DECSION

Opinion of the Institutional Ethics Committee – Please Check one
✓ Approval _____ Modifications required prior to approval (Please specify one space below) _____ Disapproval Date of review: _____ K. Thamimdha (Dr. K. MANICKAVASAKAM) Member Secretary Signed: [signature] (Please print name) Dr.V.SUBRAMANIAN Chair Person (Please delee as appropriate, Chairperson, Secretary)
Modifications needed Modification given to candidate
The research proponent is hereby informed that the Institutional Ethics Committee will require the following: 1. All adverse drug reactions (ADRs) that are both serious and unexpected to be reported promptly to the IEC within 7 working days 2. The progress report to be submitted to the IEC atleast annually 3. Upon completion of the study, a final study status report needs to submitted to the IEC

IAEC PROTOCOL NO: 1248/ac/09/CPCSEA/4-04/2011

20/12/2011

CERTIFICATE

This is certify that the project title... Preclinical and clinical Study on Azhal kalladaippu (Renal calculi) and the Drug of choice is 'Karpooram Silasathu Parpam'

has been approved by the IAEC.

Prof. Dr. K. Manickavasakam
Name of Chairman/Member Secretary IAEC:

Dr. B. Jayachandran Dare
Name of CPCSEA nominee:

Signature with date

Chairman/Member Secretary of IAEC:

CPCSEA nominee:

(Kindly make sure that minutes of the meeting duly signed by all the participants are maintained by Office)

சித்த மருத்துவ மைய ஆராய்ச்சி நிலையம், அரும்பாக்கம், சென்னை - 600 106
केन्द्रीय अनुसंधान संस्थान, अरुम्बाक्कम, चेन्नई- 600 106
Siddha Central Research Institute
Arignar Anna Govt. Hospital Campus, Arumbakkam, Chennai-600 106
(Central Council for Research in Siddha, Department of AYUSH,
Ministry of Health & Family Welfare, Govt. of India)
Phone: 044-2621 49 25
Tele Fax: 044 26214829
E-mail: crsiddha@gmail.com
Web: www.crsiddha.tn.nic.in

06.02.2012

CERTIFICATE

Certified that the minerals submitted for identification by Dr.S.Rajarajeshwari, II year Maruthuvam, National Institute of Siddha, Tambaram Sanatorium, Chennai-47 are identified as Karpoora Silasathu – Asphalt and Venkaram – Borax.

(R.Shakila)
Research Officer (Chemistry)

(K.Meenakshi Sundara Moorthy)
Asst. Director- In charge

NATIONAL INSTITUTE OF SIDDHA, CHENNAI – 600047

CERTIFICATE OF BOTANICAL AUTHENTICITY

Certified that the following plant drugs used in the Siddha formulation **Karpoora Silasathu parpam** (Internal) for the management of **Azhal Kalladaippu** taken up for Post Graduation Dissertation studies by **Dr.S.Raja Rajaeswari**, M.D.(S), II year Maruthuvam, 2011-12, are correctly identified and authenticated through Visual inspection / Organoleptic characters / Experience, Education & Training/ Morphology / Micromorphology / Microscopical/ Taxonomical methods.

Abutilon indicum (Linn.) Sw. (Malvaceae), Stem bark

Gossypium herbaceum Linn. (Malvaceae), Whole plant

Date: 10 - 1 - 12

Authorized Signatory

Dr. D. ARAVIND,
M.D (S),M.Sc. Medicinal Plants
Asst. Professor in Botany/Pharmacognosy
Dept. of Medicinal Botany
NATIONAL INSTITUTE OF SIDDHA
(DEPT. OF [illegible] GOVT. OF INDIA)
Tambaram, Chennai-600 047

SOPHISTICATED ANALYTICAL INSTRUMENT FACILITY
INDIAN INSTITUTE OF TECHNOLOGY, MADRAS
Chennai - 600 036. INDIA

CERTIFICATE

Certified that mineral drug **KARPOORA SILASTHU PARPAM** formulated by **Dr.S.RAJA RAJESHWARI** III Year M.D(S) Department of Maruthuvam, National Institute of Siddha , Tambaram Sanatorium was analysed (quantitative) by ICP-OES, HR-SEM and Physico chemical Analysis Methods at SAIF, IITM, Chennai-600 036, during October 2012.

Dr. R. MURUGESAN
Scientific Officer Gr.-I
Sophisticated Analytical Instrument Facility
Indian Institute of Technology, Madras
Chennai-600 036

Phone : 91-44-2257 4935 Fax : 91-44-2257 0545, 2257 0509
e-mail : saif@iitm.ac.in http://www.saif.iitm.ac.in

The Tamil Nadu Dr. M.G.R. Medical University

69, Anna Salai, Guindy, Chennai-600 032

This Certificate is awarded to ~~Mr/Ms~~/Dr.....S. RAJA RAJESWARI.....

for participating as a ~~Resource Person~~ / Delegate in the VII Workshop

on **"Research Methodology & Biostatistics"**

for AYUSH Post-Graduates & Researchers

organized by the Department of Siddha

The Tamil Nadu Dr. M.G.R. Medical University

from 6th Feb. 2012 to 10th Feb. 2012.

DR. MAYILVAHANAN NATARAJAN
M.S.Orth. M.Ch.Orth. (L'pool) Ph.D. (Orth. Onco.) F.R.C.S. (Eng) D.Sc.
7th VICE CHANCELLOR

Dr. R. SRILAKSHMI, DCH, Ph.D.
REGISTRAR

Dr. N. KABILAN, M.D. (Siddha)
READER, DEPT. OF SIDDHA

PATIENT USG ABDOMEN SCAN REPORT BEFORE TREATMENT

SHAKUNTHALADEVI MATERNITY HOSPITAL

NO,16,THANDAVARAYAN STREET,TONDIARPET
Chennai-600081, TEL NO:25984682

Patient name	Mrs. PADMAVATHI MARIAPPAN	**Age/Sex**	26 Years / Female
Patient ID	SD 2865	**Visit no**	1
Referred by	Dr. (SELF)	**Visit date**	26/05/2012

Whole Abdomen Scan Report

Real time B-mode Ultrasonography of Abdomen done

Abdomen

Liver normal in size and echotexture. No diffuse or focal lesion seen in the liver

IHBR not dilated.Portal vein and Hepatic veins appeared normal.CBD appeared normal.

Gall bladder distended and shows normal wall thickness. No calculi or sludge seen within the gall bladder.
Commonduct appeared normal. No calculi or sludge seen in the commonduct.
Pancreas appeared normal in size and echotexture. No ductal dilatation seen.
Spleen appeared normal.
Aorta appeared normal. No para aortic nodes seen.
Peritoneal cavity appeared normal.No Ascites.

KUB

Right kidney measured 9.4 X 2.9 cms.

Right kidney has a 4mm calculus in the lower pole with no obstructive features.

Left kidney measured 9.4 X 4.8 cms.

Cortex and collecting system of left kidney appeared normal. No calculi seen.

Bladder appeared normal.

Pelvis

Transabdominal sonography of the pelvis done
Uterus measured 8.2 X 5.3 X 6.1 cms.

Anteverted uterus with homogeneous myometrial echoes. No focal lesion seen.

Endometrial cavity appeared normal. Endometrial thickness measured 6mm.
Right ovary appeared normal and measured 3.0 x 1.8 cms in size.
Left ovary appeared normal and measured 2.8 x 1.9 cms in size.
Both adnexae appears normal.

Impression

RIGHT RENAL CALCULUS

DR. S. MAGESH

SONOLOGIST

PATIENT USG ABDOMEN SCAN REPORT AFTER TREATMENT

SHAKUNTHALADEVI MATERNITY HOSPITAL

NO,16,THANDAVARAYAN STREET,TONDIARPET

Chennai-600081, TEL NO:25984682

Patient name	Mrs. PADMAVATHI MARIYAPPAN	**Age/Sex**	34 Years / Female
Patient ID	SDM 2021	**Visit no**	1
Referred by	Dr. (SELF)	**Visit date**	24/07/2012

Whole Abdomen Scan Report

Real time B-mode Ultrasonography of Abdomen done

Abdomen

Liver normal in size and echotexture. No diffuse or focal lesion seen in the liver

IHBR not dilated.Portal vein and Hepatic veins appeared normal.CBD appeared normal.

Gall bladder distended and shows normal wall thickness. No calculi or sludge seen within the gall bladder.
Commonduct appeared normal. No calculi or sludge seen in the commonduct.
Pancreas appeared normal in size and echotexture. No ductal dilatation seen.
Spleen appeared normal.
Aorta appeared normal. No para aortic nodes seen.
Peritoneal cavity appeared normal.No Ascites.

KUB

Right kidney measured 9.0 X 4.1 cms.

Cortex and collecting system of right kidney appeared normal. No calculi seen.

Left kidney measured 9.5 X 4.7 cms.

Cortex and collecting system of left kidney appeared normal. No calculi seen.

Bladder appeared normal.

Pelvis

Transabdominal sonography of the pelvis done
Uterus measured 7.9 X 4.0 X 4.5 cms.

Anteverted uterus with homogeneous myometrial echoes. No focal lesion seen.

Endometrial cavity appeared normal. Endometrial thickness measured 8mm.
Both ovaries appear normal in size and echotexture.
Both adnexae appears normal.

Impression

* NORMAL STUDY

DR.SURESH MOHAN

SONOLOGIST

PATIENT ABDOMEN SCAN REPORT BEFORE TREATMENT

Dr.KAMAKSHI MEMORIAL HOSPITAL PVT. LTD.

Name	Mr. Karuppasamy	Date	04.07.2012
Age / Sex	38 Y/ M	ID-USG	4602
Ref. by	Dr. T. G. Govindarajan.,	Reg No	66191

USG Complete Abdomen

Liver: Size - normal Echo texture – uniform. No focal lesion. IHBR and CBD - not dilated. IVC, hepatic veins and portal vein - normal.

Gall Bladder: Distension - adequate. No calculus or internal echoes. Wall thickness - normal.

Pancreas: Size – normal. Echo texture- uniform. The pancreatic duct -not dilated.
No calcifications or peripancreatic collections.

Spleen: Size – normal. Echo texture- uniform. Long axis- 12.7 cm. No focal lesion. No hilar collaterals

Right Kidney: Position and size – normal. Size – 8.0 x 3.7 cms.
Cortical echoes -normal. CMD - maintained.
PCS - not dilated. Sinus echoes – normal. No calculus.

Left Kidney: Position and size – normal. Size – 9.6 x 6.2 cms.
Cortical echoes -normal. CMD - maintained. sinus echoes – normal.
Pcs and upper ureter appear dilated. 10mm calculus seen in left proximal ureter.

Bladder: Is normal in contour. No intraluminal echoes are seen. Wall thickness is normal.
No calculus or diverticulum is seen.

Prostate: Size – normal. Size –3.6 x 3.0 x2.9 cms. Echo texture - uniform. Volume: 15.6 cc.

RIF: Graded compression technique. No focal lesion.
No focal collection / bowel wall thickening.

No free fluid in the peritoneal cavity.

Impression:

- **Left hydroureteronephrosis due to left proximal ureteric calculus.**

-Kindly correlate clinically and with other investigations.

Dr. M. Sathyasree, MBBS, DNB.,
(Radiologist)

#1, Radial Road, Pallikaranai, Chennai - 600 100. Tel : 044 - 6630 0300, 2246 9200
Fax : 044 - 2246 3282, 6630 0400 E-mail : info@drkmh.com Website : www.drkmh.com

PATIENT USG ABDOMEN SCAN REPORT AFTER TREATMENT

INDIAN SCAN
ADVANCED DIAGNOSTIC CENTRE

• Multi Channel MRI • Multi Slice 3D Spiral CT • Digital Color Doppler • Digital Ultrasonography • Echocardiography
• Computerised ECG • Treadmill • PFT • Digital X-Ray • Laboratory • Sonomammography • 4D Scan • EEG • Digital Mammogram

Patient : MR.KARUPU SAMY
Ref.By : DR.RAJA RAJESWARI.,
Age/Sex : 38 yrs / M
Date : 02.08.2012

ULTRASONOGRAPHY REPORT – ABDOMEN / MALE

LIVER :
Normal in size and echo pattern. No focal or diffuse pathology.
CBD and IHBR appear normal. Portal vein is normal.

GALL BLADDER :
Normal in size. Wall is normal.
No calculus / sludge / polyp.

PANCREAS :
Normal in size & echo pattern. Pancreatic duct is not dilated.
No focal / diffuse pathology.

SPLEEN :
Normal in size and echo pattern.

KIDNEYS :
Right kidney appears contracted and it measures 80.7 x 34.9 mm.
Cortical echoes are normal.
Collecting system is normal. No evidence of calculus.

Left kidney measures 101.4 x 56.7 mm.
Cortical echoes are normal. No focal lesion.
Collecting system is normal. No evidence of calculus.

URINARY BLADDER:
Distended. Wall is normal. Bladder wall thickness 3.5 mm.
No abnormal intraluminal echoes.

PROSTATE :
Prostate appears normal .
It measures 40.8 x 28.6 x 26.4 mm. Wt . 16.1 gms.
No focal lesion.

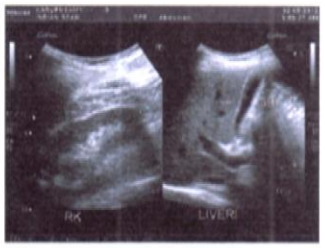
PERITONEUM:
No evidence of ascites.

AORTIC & IVC:
Normal in calibre. No demonstrable para aortic nodes.

RIGHT ILIAC FOSSA:
No ultra sonographically demonstrable pathology or tenderness.

IMPRESSION :

CONTRACTED RIGHT KIDNEY



DR. G.NIRMAL KUMAR, M.D.,R.D.,
Consultant Radiologist

No. 7/9, Duraisamy Pillai Street, West Tambaram, Chennai - 45. Ph : 22262428, 22261473

24 HOURS EMERGENCY SERVICE ◆ AMBULANCE SERVICE AVAILABLE ON REQUEST

PATIENT URINE CULTURE AND SENSITIVITY BEFORE TREATMENT

LAB REPORT

PARVATHY HOSPITAL
FUTURISTIC ORTHO & NEURO

Name	: MrsAMUDHA V		
Age / Gender	: 37 Years /Female	IP No.	: IPC1.0014701
Requisition No.	: 167107	Reporting Date	: 31/Aug/2012 03:24:17 PM
UHID Number	: POHS.0000078126	Result Status	: Result Verified
Referred By	: Dr MUTHU KUMAR AND TEAM	Print Date	: 1/9/2012 4:36:50PM

URINE CULTURE

URINE CULTURE

NAME OF SPECIMEN : URINE

NAME OF ORGANISM : E-COLI GROWN IN CULTURE

COLONY COUNT : >100,000 org/ml

GRAM STAIN : GRAM NEGATIVE BACILLI

HIGHLY SENSITIVE TO : POLYMYXIN,LEVOFLOXACIN,GENTAMYCIN
CEPHOTAXIME,COTRIMOXAZOLE,
MEROPENEM,CIPROFLOXACIN,CEFIXIME

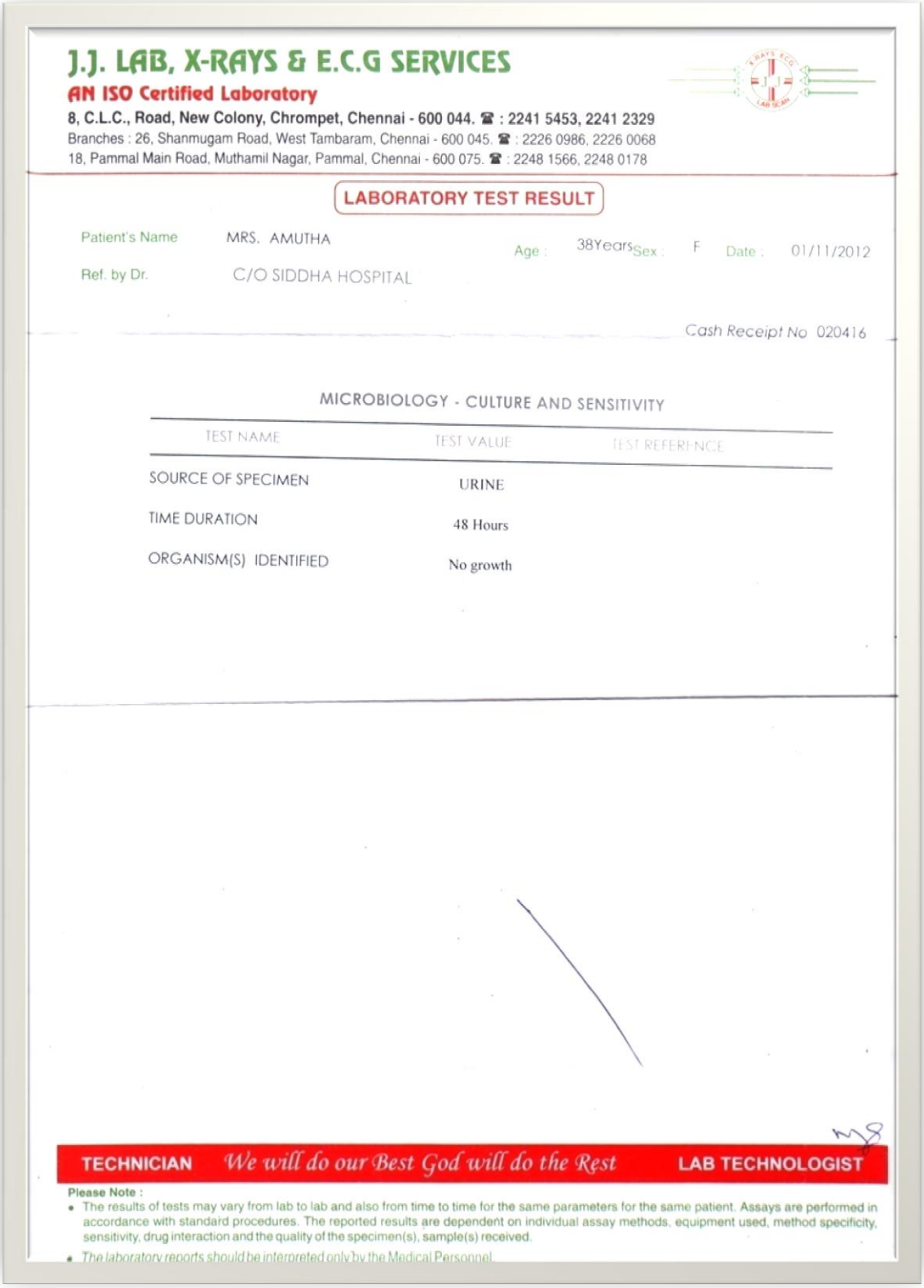
MODERATE SENSITIVE TO : AMPICILLIN/SULBACTUM,CEFTRIAXONE

RESISTANT TO : AMOXICILLIN/CLAVULANIC ACID
CEFOPEROZONE/SULBACTUM

Sign :
Name :
(Authorised Doctor)

Department of Clinical Pathology | Department of Bio-Chemistry | Department of Microbiology | Department of Pathology | Laboratory Chief Technician

PATIENT URINE CULTURE AND SENSITIVITY AFTER TREATMENT



J.J. LAB, X-RAYS & E.C.G SERVICES

AN ISO Certified Laboratory

8, C.L.C., Road, New Colony, Chrompet, Chennai - 600 044. ☎ : 2241 5453, 2241 2329
Branches : 26, Shanmugam Road, West Tambaram, Chennai - 600 045. ☎ : 2226 0986, 2226 0068
18, Pammal Main Road, Muthamil Nagar, Pammal, Chennai - 600 075. ☎ : 2248 1566, 2248 0178

LABORATORY TEST RESULT

Patient's Name MRS. AMUTHA Age : 38Years Sex : F Date : 01/11/2012

Ref. by Dr. C/O SIDDHA HOSPITAL

Cash Receipt No 020416

MICROBIOLOGY - CULTURE AND SENSITIVITY

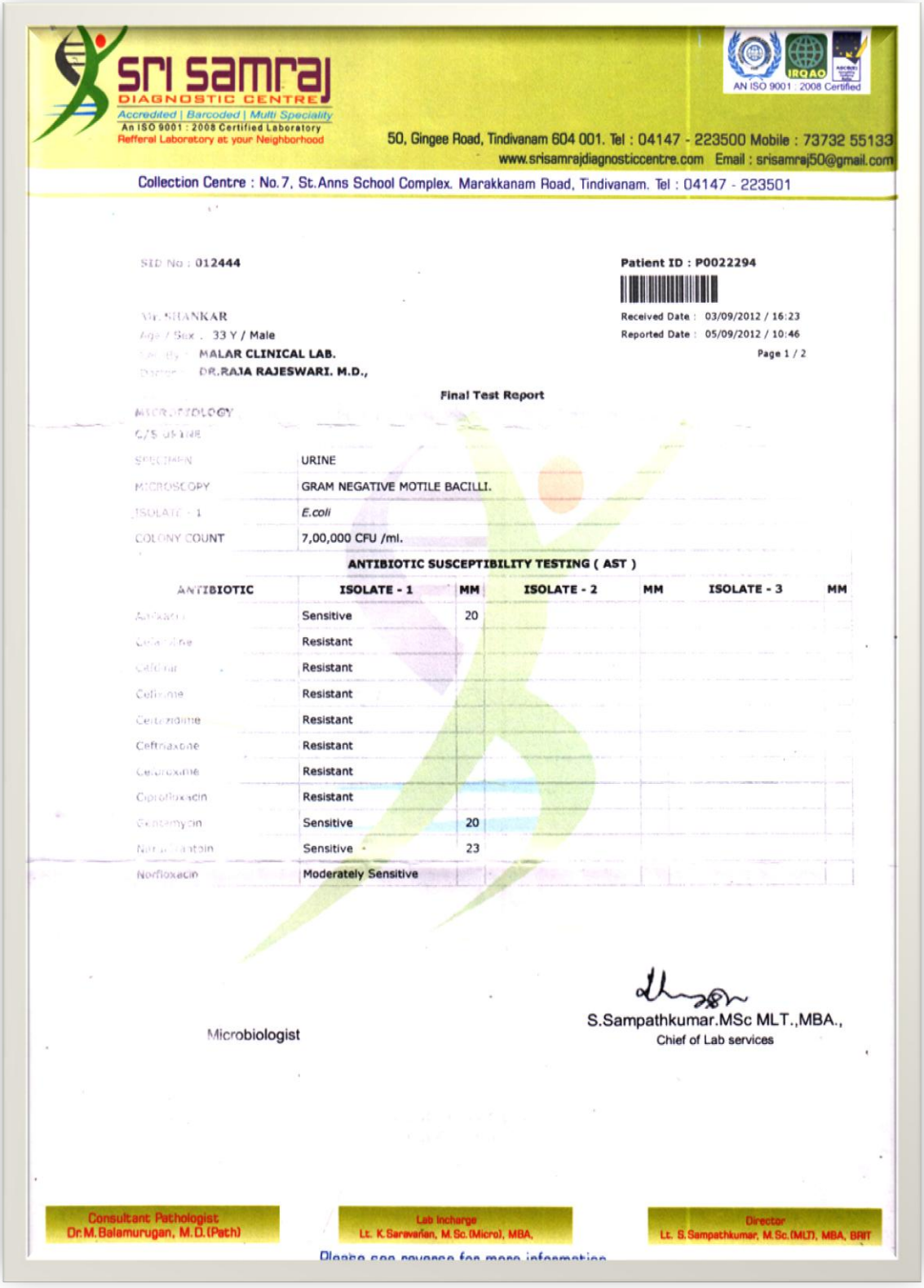
TEST NAME	TEST VALUE	TEST REFERENCE
SOURCE OF SPECIMEN	URINE	
TIME DURATION	48 Hours	
ORGANISM(S) IDENTIFIED	No growth	

TECHNICIAN *We will do our Best God will do the Rest* LAB TECHNOLOGIST

Please Note :

- The results of tests may vary from lab to lab and also from time to time for the same parameters for the same patient. Assays are performed in accordance with standard procedures. The reported results are dependent on individual assay methods, equipment used, method specificity, sensitivity, drug interaction and the quality of the specimen(s), sample(s) received.
- The laboratory reports should be interpreted only by the Medical Personnel.

PATIENT URINE CULTURE AND SENSITIVITY BEFORE TREATMENT



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50, Gingee Road, Tindivanam 604 001. Tel : 04147 - 223500 Mobile : 73732 55133
www.srisamrajdiagnosticcentre.com Email : srisamraj50@gmail.com

Collection Centre : No.7, St.Anns School Complex. Marakkanam Road, Tindivanam. Tel : 04147 - 223501

SID No : 012444

Patient ID : P0022294

Mr.SHANKAR
Age / Sex : 33 Y / Male
Society : MALAR CLINICAL LAB.
Doctor : DR.RAJA RAJESWARI. M.D.,

Received Date : 03/09/2012 / 16:23
Reported Date : 05/09/2012 / 10:46
Page 1 / 2

Final Test Report

MICROBIOLOGY

C/S URINE

SPECIMEN	URINE
MICROSCOPY	GRAM NEGATIVE MOTILE BACILLI.
ISOLATE - 1	*E.coli*
COLONY COUNT	7,00,000 CFU /ml.

ANTIBIOTIC SUSCEPTIBILITY TESTING (AST)

ANTIBIOTIC	ISOLATE - 1	MM	ISOLATE - 2	MM	ISOLATE - 3	MM
Amikacin	Sensitive	20				
Cefadroxil	Resistant					
Cefotaxime	Resistant					
Cefixime	Resistant					
Ceftazidime	Resistant					
Ceftriaxone	Resistant					
Cefuroxime	Resistant					
Ciprofloxacin	Resistant					
Gentamycin	Sensitive	20				
Nitrofurantoin	Sensitive	23				
Norfloxacin	Moderately Sensitive					

Microbiologist

S.Sampathkumar.MSc MLT.,MBA.,
Chief of Lab services

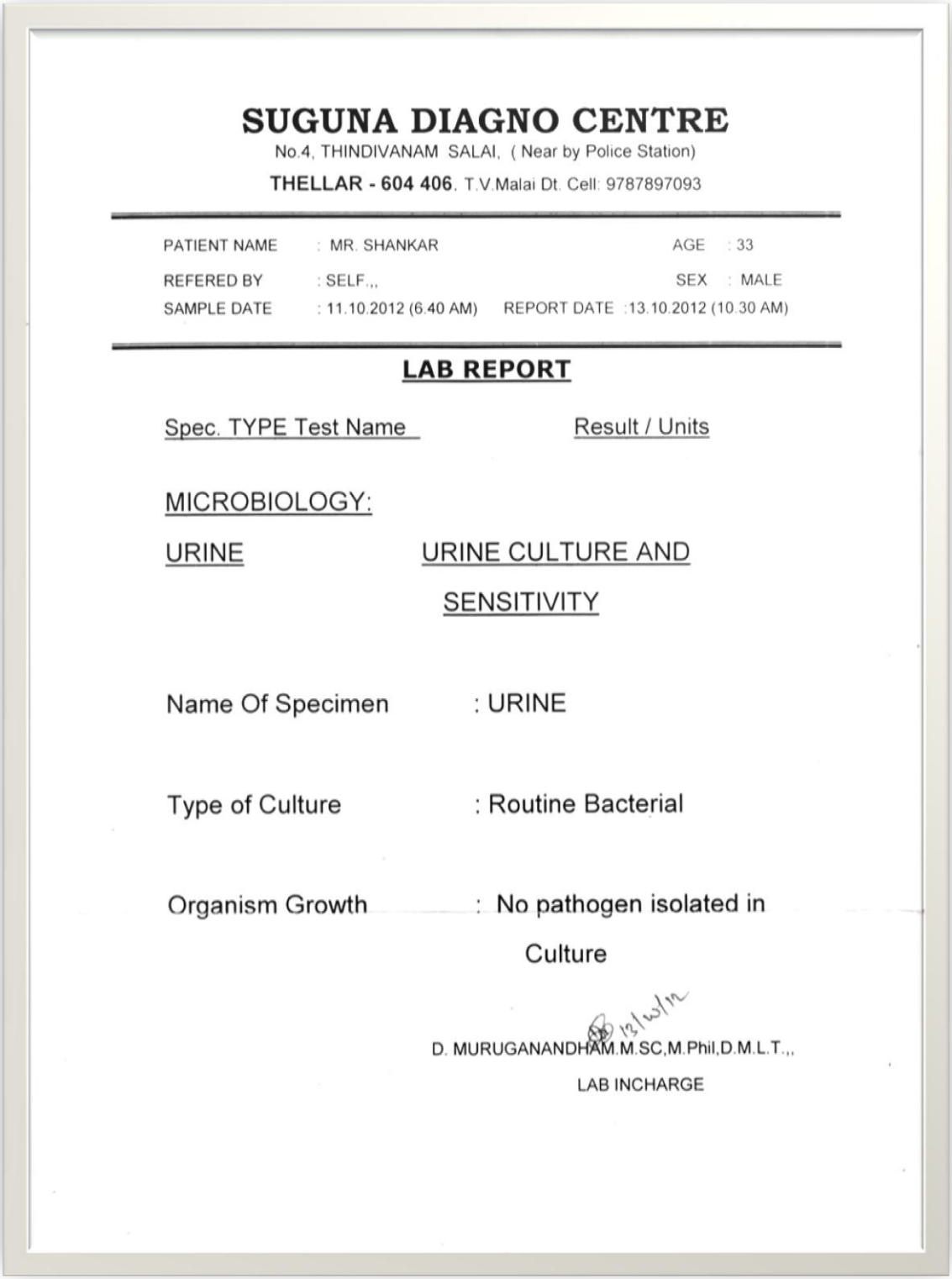
Consultant Pathologist
Dr.M.Balamurugan, M.D.(Path)

Lab Incharge
Lt. K.Saravanan, M.Sc.(Micro), MBA.

Director
Lt. S.Sampathkumar, M.Sc.(MLT), MBA, BRT

Please see reverse for more information

PATIENT URINE CULTURE AND SENSITIVITY AFTER TREATMENT



SUGUNA DIAGNO CENTRE

No.4, THINDIVANAM SALAI, (Near by Police Station)

THELLAR - 604 406, T.V.Malai Dt. Cell: 9787897093

PATIENT NAME	: MR. SHANKAR	AGE	: 33
REFERED BY	: SELF.,,	SEX	: MALE
SAMPLE DATE	: 11.10.2012 (6.40 AM)	REPORT DATE	:13.10.2012 (10.30 AM)

LAB REPORT

Spec. TYPE Test Name	Result / Units
MICROBIOLOGY:	
URINE	URINE CULTURE AND SENSITIVITY
Name Of Specimen	: URINE
Type of Culture	: Routine Bacterial
Organism Growth	: No pathogen isolated in Culture

13/10/12

D. MURUGANANDHAM.M.SC,M.Phil,D.M.L.T.,,

LAB INCHARGE

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2. agathiyar gunavagadam
3. agathiyar paripooranam – 400
4. agathiyar naadi
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6. dhanvanthiri vaithya kaviyam
7. jeevaratchamirtham
8. siddha maruthuvam noi vilakkam
9. sattamuni gnanam
10. sathaga naadi
11. thirumoolar 800
12. thirumoolar karukadai vaithiyam
13. theran karisal
14. theraiyar gunavagadam
15. tamil vaithyasathagam
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CLINICAL ASSESSMENTS

Sl. No	OP/ IP NO	Age/Sex	Abdominal pain		Painfrom loin to groin region		Abdominal distension		Pain in urethra		Agonizing pain		dysuria		oliguria		Burning micturition		Yellow coloured urination		Nausea/ vomiting		Haematuria	
			BT	AT	BT	AT	BT	AT	BT	AT	BT	AT	BT	AT	BT	AT	BT	AT	BT	AT	BT	AT	BT	AT
1	C69642	24/F	+	-	+	-	-	-	-	-	-	-	+	-	+	-	+	-	-	-	+	-	-	-
2	C71045	31/M	+	-	+	-	-	-	+	-	+	-	-	-	+	-	+	-	+	-	-	-	-	-
3	C73030	43/M	+	-	+	-	-	-	+	-	+	-	-	-	+	-	+	-	+	-	-	-	-	-
4	C73050	43/M	+	-	+	-	-	-	+	-	+	-	-	-	+	-	+	-	+	-	+	-	-	-
5	C72496	32/F	+	-	+	-	-	-	+	-	+	-	+	-	+	-	+	-	+	-	-	-	-	-
6	C74152	52/M	+	-	+	-	-	-	+	-	+	-	+	-	+	-	+	-	+	-	-	-	-	-
7	C73299	32/M	+	-	+	-	-	-	+	-	+	-	-	-	+	-	+	-	+	-	-	-	-	-
8	C73332	42/M	+	-	+	-	-	-	+	-	+	-	+	-	+	-	+	-	+	-	+	-	-	-
9	C75054	50/F	+	-	+	-	-	-	-	-	-	-	-	-	+	-	+	-	+	-	-	-	-	-
10	C76543	24/M	+	-	+	-	-	-	-	-	-	-	-	-	+	-	+	-	+	-	-	-	-	-
11	C74751	33/M	+	-	+	-	-	-	-	-	-	-	-	-	+	-	+	-	+	-	-	-	-	-
12	C77242	34/M	+	-	+	-	-	-	-	-	-	-	-	-	+	-	+	-	+	-	-	-	-	-
13	C77701	28/M	+	-	+	-	-	-	-	-	+	-	+	-	+	-	+	-	+	-	-	-	-	-
14	C78713	53/F	+	-	+	-	-	-	-	-	-	-	-	-	+	-	+	-	+	-	-	-	-	-
15	C79434	29/F	+	-	+	-	-	-	-	-	-	-	-	-	+	-	-	-	-	-	-	-	-	-
16	C78007	38/M	+	-	+	-	-	-	-	-	-	-	+	-	+	-	+	-	+	-	-	-	-	-
17	C80489	40/M	+	-	+	-	-	-	-	-	-	-	-	-	-	-	-	-	+	-	-	-	-	-
18	C80919	29/F	+	-	+	-	-	-	-	-	-	-	-	-	+	-	-	-	+	-	-	-	-	-
19	C81353	60/M	+	-	+	-	-	-	-	-	-	-	+	-	+	-	+	-	+	-	-	-	-	-
20	C81148	36/M	+	-	+	-	-	-	-	-	-	-	-	-	-	-	-	-	+	-	-	-	-	-

Sl. No	OP/ IP NO	Age/Sex	Abdominal pain		Painfrom loin to groin region		Abdominal distension		Pain in urethra		Agonizing pain		dysuria		oliguria		Burning micturition		Yellow coloured urination		Nausea/ vomiting		Haematuria	
			BT	AT	BT	AT	BT	AT	BT	AT	BT	AT	BT	AT	BT	AT	BT	AT	BT	AT	BT	AT	BT	AT
21	C82243	22/M	+	-	+	-	-	-	-	-	-	-	-	-	-	-	-	-	+	-	-	-	-	-
22	C81712	60/M	+	-	+	-	-	-	-	-	-	-	-	-	-	-	-	-	+	-	-	-	-	-
23	C82707	42/M	+	-	+	-	-	-	-	-	-	-	-	-	+	-	-	-	-	-	-	-	-	-
24	C82990	27/M	+	-	+	-	-	-	-	-	-	-	+	-	-	-	-	-	-	-	-	-	-	-
25	C84030	38/M	+	-	+	-	-	-	-	-	-	-	+	-	+	-	+	-	+	-	-	-	-	-
26	C80753	48/M	+	-	+	-	-	-	-	-	-	-	-	-	+	-	-	-	-	-	-	-	-	-
27	C85766	32/M	+	-	+	-	-	-	-	-	-	-	-	-	+	-	+	-	+	-	-	-	-	-
28	C85523	25/M	+	-	+	-	-	-	-	-	-	-	-	-	-	-	+	-	+	-	-	-	-	-
29	C87868	42/F	+	-	+	-	-	-	-	-	-	-	-	-	-	-	+	-	-	-	-	-	-	-
30	C86025	40/M	+	-	+	-	-	-	-	-	-	-	-	-	-	-	-	-	+	-	-	-	-	-
31	C88682	45/M	+	-	+	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-
32	C89476	33/F	+	-	+	-	-	-	-	-	-	-	-	-	+	-	+	-	+	-	-	-	-	-
33	C95276	37/F	+	-	+	-	-	-	-	-	-	-	-	-	-	-	+	-	-	-	-	-	-	-
34	3888	52/F	+	-	+	-	-	-	-	-	-	-	-	-	+	-	+	-	+	-	-	-	-	-
35	4965	32/M	+	-	+	-	-	-	-	-	-	-	-	-		-	+	-	+	-	-	-	-	-
36	5055	48/M	+	-	+	-	-	-	-	-	-	-	+	-	+	-	+	-	+	-	-	-	-	-
37	4061	32/F	+	-	+	-	-	-	-	-	-	-	-	-	-	-	-	-	+	-	+	-	-	-
38	4099	35/F	+	-	+	-	-	-	-	-	-	-	-	-	+	-	-	-	-	-	-	-	-	-
39	4156	55/F	+	-	+	-	-	-	-	-	-	-	-	-	-	-	+	-	-	-	-	-	-	-
40	5115	27/M	+	-	+	-	-	-	-	-	-	-	-	-	-	-	+	-	+	-	-	-	-	-

BT - Before Treatment AT – After Treatment

URINE AND MOTION INVESTIGATIONS

S. NO	OP/IP NO	URINE													MOTION					
		Before Treatment							After Treatment							Before Treatment			After Treatment		
		Alb	Sug	Deposits		BS	BP	URO	Alb	Sug	Deposits		BS	BP	URO	Ova	Cyst	Occult bld	Ova	Cyst	Occult blood
				Pus Cells	Epi. cells						Pus cells	Epi. cells									
1.	C69642	NIL	NIL	4-5	4-5	NIL	(-)ve	normal	NIL	NIL	1-2	1-2	NIL	(-)ve	normal	NIL	NIL	NIL	NIL	NIL	NIL
2.	C71045	NIL	NIL	8-10	5-6	NIL	(-)ve	normal	NIL	NIL	1-2	1-2	NIL	(-)ve	normal	NIL	NIL	NIL	NIL	NIL	NIL
3.	C73030	NIL	NIL	4-5	4-5	NIL	(-)ve	normal	NIL	NIL	1-2	1-2	NIL	(-)ve	normal	NIL	NIL	NIL	NIL	NIL	NIL
4.	C73050	NIL	NIL	1-2	2-3	NIL	(-)ve	normal	NIL	NIL	2-3	2-3	NIL	(-)ve	normal	NIL	NIL	NIL	NIL	NIL	NIL
5.	C72496	NIL	NIL	4-5	4-5	NIL	(-)ve	normal	NIL	NIL	1-2	2-4	NIL	(-)ve	normal	NIL	NIL	NIL	NIL	NIL	NIL
6.	C74152	NIL	NIL	2-3	1-2	NIL	(-)ve	normal	NIL	NIL	2-3	2-3	NIL	(-)ve	normal	NIL	NIL	NIL	NIL	NIL	NIL
7.	C73299	NIL	NIL	3-4	3-4	NIL	(-)ve	normal	NIL	NIL	1-2	1-2	NIL	(-)ve	normal	NIL	NIL	NIL	NIL	NIL	NIL
8.	C73332	NIL	NIL	1-2	2-3	NIL	(-)ve	normal	NIL	NIL	2-4	2-4	NIL	(-)ve	normal	NIL	NIL	NIL	NIL	NIL	NIL
9.	C75054	NIL	NIL	1-2	1-2	NIL	(-)ve	normal	NIL	NIL	2-4	2-4	NIL	(-)ve	normal	NIL	NIL	NIL	NIL	NIL	NIL
10.	C76543	NIL	NIL	2-4	2-4	NIL	(-)ve	normal	NIL	NIL	1-2	1-2	NIL	(-)ve	normal	NIL	NIL	NIL	NIL	NIL	NIL
11.	C74751	NIL	NIL	1-2	1-2	NIL	(-)ve	normal	NIL	NIL	1-2	1-2	NIL	(-)ve	normal	NIL	NIL	NIL	NIL	NIL	NIL
12.	C77242	NIL	NIL	1-2	1-2	NIL	(-)ve	normal	NIL	NIL	1-2	1-2	NIL	(-)ve	normal	NIL	NIL	NIL	NIL	NIL	NIL
13.	C77701	NIL	NIL	1-2	1-2	NIL	(-)ve	normal	NIL	NIL	2-4	2-4	NIL	(-)ve	normal	NIL	NIL	NIL	NIL	NIL	NIL
14.	C78713	NIL	NIL	2-3	2-3	NIL	(-)ve	normal	NIL	NIL	1-2	1-2	NIL	(-)ve	normal	NIL	NIL	NIL	NIL	NIL	NIL
15.	C79434	NIL	NIL	2-3	2-3	NIL	(-)ve	normal	NIL	NIL	2-4	1-2	NIL	(-)ve	normal	NIL	NIL	NIL	NIL	NIL	NIL
16.	C78007	NIL	NIL	1-2	1-2	NIL	(-)ve	normal	NIL	NIL	2-3	3-5	NIL	(-)ve	normal	NIL	NIL	NIL	NIL	NIL	NIL
17.	C80489	NIL	NIL	2-4	2-4	NIL	(-)ve	normal	NIL	NIL	1-2	1-2	NIL	(-)ve	normal	NIL	NIL	NIL	NIL	NIL	NIL
18.	C80919	NIL	NIL	1-2	2-3	NIL	(-)ve	normal	NIL	NIL	2-4	2-4	NIL	(-)ve	normal	NIL	NIL	NIL	NIL	NIL	NIL
19.	C81353	NIL	NIL	1-2	1-2	NIL	(-)ve	normal	NIL	NIL	2-3	1-2	NIL	(-)ve	normal	NIL	NIL	NIL	NIL	NIL	NIL
20.	C81148	NIL	NIL	2-4	2-4	NIL	(-)ve	normal	NIL	NIL	1-2	1-2	NIL	(-)ve	normal	NIL	NIL	NIL	NIL	NIL	NIL

URINE AND MOTION INVESTIGATIONS

S. NO	OP/IP NO	URINE														MOTION					
		Before Treatment							After Treatment							Before Treatment			After Treatment		
		Alb	Sug	Deposits		BS	BP	URO	Albumin	Sugar	Deposits		BS	BP	URO	Ova	Cyst	Occult bld	Ova	Cyst	Occult blood
				Pus Cells	Epi. Cells						Pus Cells	Epi. Cells									
21.	C82243	NIL	NIL	1-2	2-3	NIL	(-)ve	normal	NIL	NIL	2-3	1-2	NIL	(-)ve	normal	NIL	NIL	NIL	NIL	NIL	NIL
22.	C81712	NIL	NIL	2-4	3-5	NIL	(-)ve	normal	NIL	NIL	3-4	3-4	NIL	(-)ve	normal	NIL	NIL	NIL	NIL	NIL	NIL
23.	C82707	NIL	NIL	2-4	3-5	NIL	(-)ve	normal	NIL	NIL	2-4	3-5	NIL	(-)ve	normal	NIL	NIL	NIL	NIL	NIL	NIL
24.	C82990	NIL	NIL	2-4	1-2	NIL	(-)ve	normal	NIL	NIL	2-4	1-2	NIL	(-)ve	normal	NIL	NIL	NIL	NIL	NIL	NIL
25.	C84030	NIL	NIL	2-3	1-2	NIL	(-)ve	normal	NIL	NIL	1-2	1-2	NIL	(-)ve	normal	NIL	NIL	NIL	NIL	NIL	NIL
26.	C80753	NIL	NIL	1-2	2-4	NIL	(-)ve	normal	NIL	NIL	1-2	1-2	NIL	(-)ve	normal	NIL	NIL	NIL	NIL	NIL	NIL
27.	C85766	NIL	NIL	1-2	2-3	NIL	(-)ve	normal	NIL	NIL	2-4	1-2	NIL	(-)ve	normal	NIL	NIL	NIL	NIL	NIL	NIL
28.	C85523	NIL	NIL	2-4	2-4	NIL	(-)ve	normal	NIL	NIL	2-3	2-3	NIL	(-)ve	normal	NIL	NIL	NIL	NIL	NIL	NIL
29.	C87868	NIL	NIL	1-3	2-3	NIL	(-)ve	normal	NIL	NIL	3-4	2-3	NIL	(-)ve	normal	NIL	NIL	NIL	NIL	NIL	NIL
30.	C86025	NIL	NIL	4-5	4-5	NIL	(-)ve	normal	NIL	NIL	2-4	2-4	NIL	(-)ve	normal	NIL	NIL	NIL	NIL	NIL	NIL
31.	C88682	NIL	NIL	2-4	3-5	NIL	(-)ve	normal	NIL	NIL	2-3	2-3	NIL	(-)ve	normal	NIL	NIL	NIL	NIL	NIL	NIL
32.	C89476	NIL	NIL	1-2	1-2	NIL	(-)ve	normal	NIL	NIL	1-2	1-2	NIL	(-)ve	normal	NIL	NIL	NIL	NIL	NIL	NIL
33.	C95276	NIL	NIL	4-5	4-5	NIL	(-)ve	normal	NIL	NIL	2-3	2-3	NIL	(-)ve	normal	NIL	NIL	NIL	NIL	NIL	NIL
34.	3888	NIL	NIL	1-2	1-2	NIL	(-)ve	normal	NIL	NIL	1-2	1-2	NIL	(-)ve	normal	NIL	NIL	NIL	NIL	NIL	NIL
35.	4965	NIL	NIL	3-4	1-2	NIL	(-)ve	normal	NIL	NIL	1-2	1-2	NIL	(-)ve	normal	NIL	NIL	NIL	NIL	NIL	NIL
36.	5055	NIL	NIL	2-4	2-4	NIL	(-)ve	normal	NIL	NIL	1-2	2-4	NIL	(-)ve	normal	NIL	NIL	NIL	NIL	NIL	NIL
37.	4061	NIL	NIL	1-2	1-2	NIL	(-)ve	normal	NIL	NIL	1-2	1-2	NIL	(-)ve	normal	NIL	NIL	NIL	NIL	NIL	NIL
38.	4099	NIL	NIL	1-2	2-3	NIL	(-)ve	normal	NIL	NIL	1-2	1-2	NIL	(-)ve	normal	NIL	NIL	NIL	NIL	NIL	NIL
39.	4156	NIL	NIL	1-2	1-2	NIL	(-)ve	normal	NIL	NIL	2-3	1-2	NIL	(-)ve	normal	NIL	NIL	NIL	NIL	NIL	NIL
40.	5115	NIL	NIL	1-2	2-3	NIL	(-)ve	normal	NIL	NIL	1-2	1-2	NIL	(-)ve	normal	NIL	NIL	NIL	NIL	NIL	NIL

LAB INVESTIGATIONS

Hb and RBC – INVESTIGATIONS BEFORE AND AFTER TREATMENT – IN OP & IP PATIENTS

SL. NO	OP/IP NO	NAME	Age/sex	Hbgm%		TRBC Million cells /cumm	
				Before treatment	After treatment	Before treatment	After treatment
1.	C69642	Mrs.P.Sangeetha	24/F	12.9	14.3	4.4	4.9
2.	C71045	Mr.B.Muthusaravanan	31/M	14.2	14.3	5.0	5.0
3.	C73030	Mr.D.Vijayakumar	43/M	17.4	16.4	5.5	5.3
4.	C73050	Mr.E.Esakkimuthu	43/M	15.0	15.4	5.5	5.5
5.	C72496	Mrs.M.Padmavathy	32/F	12.0	11.7	4.4	4.5
6.	C74152	Mr.R.Radhakrishnan	52/M	9.7	10.1	4.0	4.1
7.	C73299	Mr.M.Tamizlselvan	32/M	16.1	15.9	5.2	5.3
8.	C73332	Mr.S.Gothandapani	42/M	15.2	16.1	4.8	5.2
9.	C75054	Mrs.R.Shanthi	50/F	13.0	12.9	4.5	4.3
10.	C76543	Mr.M.Dillibabu	24/M	15.5	15.7	4.9	4.9
11.	C74751	Mr.S.V.anjuman	33/M	16.3	17.3	6.0	6.2
12.	C77242	Mr.N.Vijayakumar	34/M	15.4	15.7	5.0	5.1
13.	C77701	Mr.D.Harish	28/M	12.5	15.5	3.9	4.9
14.	C78713	Mrs.K.Chandra	53/F	13.4	12.9	4.8	4.4
15.	C79434	Msr.V.Logeswari	29/F	12.2	11.8	4.5	4.5
16.	C78007	Mr.B.Raj	38/M	16.0	15.4	5.5	5.2
17.	C80489	Mr.S.Mani	40/M	14.8	15.2	5.5	5.5
18.	C80919	Mrs.S.Amudha	29/F	12.1	12.6	4.1	4.2
19.	C81353	Mr.I.Selvaraj	60/M	14.9	15.0	5.1	5.1
20.	C81148	Mr.R.Baburajan	36/M	15.3	15.8	5.5	5.7
21.	C82243	Mr.J.Chandramohan	22/M	17.2	17.6	5.5	5.6
22.	C81712	Mr.P.Ramadoss	60/M	13.7	14.1	4.5	4.7
23.	C82707	Mr.R.Sachithanantham	42/M	13.9	14.6	4.9	5.2
24.	C82990	Mr.M.Karthick	27/M	16.7	15.8	5.5	5.2
25.	C84030	Mr.D.Karuppasamy	38/M	15.4	15.0	4.9	4.7
26.	C80753	Mr.K.Ravichandiran	48/M	14.6	14.9	4.8	5.0
27.	C85766	Mr.P.Anthonydoss	32/M	16.4	15.4	5.8	5.4
28.	C85523	Mr.S.Raja	25/M	15.8	14.6	5.4	4.9
29.	C87868	Mrs.R.Sumathy	42/F	13.9	13.5	4.7	4.5
30.	C86025	Mr.A.Raman	40/M	15.6	13.8	5.6	5.7
31.	C88682	Mr.C.Kumar	45/M	14.3	14.6	4.8	4.9
32.	C89476	Mr.M.Shankar	33/F	16.5	16.6	5.4	5.6
33.	C95276	Mrs.V.Amudha	37/F	12.6	13.8	4.0	4.0
34.	3888	Mrs.K.Kanaga	52/F	17.4	16	5.8	5.9
35.	4965	M.Sathyamoorthy	32/M	15.2	14.8	4.9	4.6
36.	5055	Mr.M.Gopalsamy	48/M	15.3	15.2	4.3	4.1
37.	4061	Mrs.R.Devi	32/F	10.7	11.7	4.1	4.0
38.	4099	Mrs.N.Anjali	35/F	13.8	13.8	4.3	4.4
39.	4156	Mrs.R.Nalini	55/F	10.3	11.6	3.6	3.9
40.	5115	Mr.S.Akbarali	27/M	15.1	15.6	4.4	4.9

LAB INVESTIGATIONS BEFORE TREATMENT

Sno	OP/IP No	Age/Sex	TC Cells/ cumm	DC%			ESR mm/hr		Blood sugar mg/dl		Blood urea mg/dl	S.Creatinine Mg/dl	S.Uric acid mg/dl
				P	L	E	1/2 hr	1 hr	F	PP			
1	C69642	24/F	10300	62	28	10	4	10	107	129	19	0.6	6.1
2	C71045	31/M	9800	60	28	2	4	8	95	114	18	0.6	6.4
3	C73030	43/M	6900	62	35	3	2	6	115	123	28	0.6	7.2
4	C73050	43/M	6900	63	34	3	2	4	79	101	22	0.7	5.5
5	C72496	32/F	4900	62	36	2	8	16	86	102	21	0.6	4.9
6	C74152	52/M	7500	64	32	4	12	18	102	112	37	0.8	6.0
7	C73299	32/M	4000	56	30	4	2	4	94	92	30	0.7	5.4
8	C73332	42/M	9500	43	32	5	4	8	104	100	33	0.8	8.2
9	C75054	50/F	8600	55	34	4	8	22	86	125	15	0.6	4.7
10	C76543	24/M	6400	64	30	6	2	4	84	99	19	0.6	6.0
11	C74751	33/M	7200	54	40	6	2	6	81	102	23	0.7	6.5
12	C77242	34/M	5200	69	25	1	2	6	104	114	18	0.5	5.4
13	C77701	28/M	4500	50	34	6	4	8	96	86	17	0.5	7.9
14	C78713	53/F	8600	50	32	6	4	12	68	121	19	0.5	5.2
15	C79434	29/F	6700	48	34	2	2	6	104	128	16	0.5	4.4
16	C78007	38/M	9900	59	34	7	4	10	95	107	17	0.5	6.6
17	C80489	40/M	7600	22	36	3	2	12	104	128	21	0.6	6.8
18	C80919	29/F	7400	68	30	2	6	16	71	110	20	0.6	3.5
19	C81353	60/M	7000	64	30	6	4	8	100	108	23	0.8	7.0
20	C81148	36/M	7400	49	36	5	2	4	93	119	28	0.8	3.7
21	C82243	22/M	5800	54	20	5	2	4	84	103	23	0.7	5.5
22	C81712	60/M	5500	65	30	5	2	4	127	138	21	0.7	6.0
23	C82707	42/M	7800	45	39	6	2	6	85	105	18	0.6	6.7
24	C82990	27/M	7300	64	30	5	2	4	96	107	20	0.8	7.4
25	C84030	38/M	7200	47	34	6	2	4	82	123	22	0.6	5.9
26	C80753	48/M	5900	60	34	6	6	14	101	111	23	0.8	5.3
27	C85766	32/M	8800	48	37	1	2	4	89	121	16	0.5	6.0
28	C85523	25/M	10700	66	30	4	10	20	88	112	22	0.7	5.5
29	C87868	42/F	9800	67	30	3	4	10	107	110	15	0.6	2.7
30	C86025	40/M	7400	52	30	8	2	6	99	103	19	0.6	4.9
31	C88682	45/M	5700	54	33	6	2	4	110	125	15	0.5	5.7
32	C89476	33/F	7800	62	35	3	2	4	86	104	19	0.5	4.5
33	C95276	37/F	8300	62	34	4	3	8	99	109	18	0.6	4.5
34	3888	52/F	8600	55	38	5	6	12	70	89	25	0.8	5.8
35	4965	32/M	5100	53	29	6	2	4	80	100	19	0.5	4.1
36	5055	48/M	7600	65	30	5	2	4	85	100	14	0.4	5.6
37	4061	32/F	11000	73	21	6	6	12	93	103	15	0.6	6.9
38	4099	35/F	10500	56	31	2	6	18	94	118	17	0.5	3.1
39	4156	55/F	8500	50	40	2	12	24	88	103	23	0.6	3.0
40	5115	27/M	6500	46	28	5	4	12	83	99	15	0.4	4.6

LAB INVESTIGATIONS AFTER TREATMENT

Sno	OP/IP No	Age/Sex	TC Cells/ cumm	DC%			ESR mm/hr		Blood sugar mg/dl		Blood urea mg/dl	S.creatinine Mg/dl	S.Uric acid mg/dl
				P	L	E	1/2 hr	1 hr	F	PP			
1	C69642	24/F	9900	67	30	3	2	6	92	113	30	0.9	6.0
2	C71045	31/M	9300	66	27	5	2	4	98	109	19	0.6	5.0
3	C73030	43/M	7700	55	30	5	6	14	103	109	27	0.7	5.9
4	C73050	43/M	6800	48	22	1	4	8	71	98	12	0.4	4.7
5	C72496	32/F	5300	65	26	8	6	12	102	108	15	0.5	4.5
6	C74152	52/M	7400	69	25	6	12	24	104	111	41	1.1	3.0
7	C73299	32/M	3700	46	27	7	2	6	84	93	22	0.7	4.7
8	C73332	42/M	9000	55	39	6	2	4	83	103	15	0.5	6.0
9	C75054	50/F	8900	60	35	5	2	4	115	121	15	0.4	5.3
10	C76543	24/M	6700	63	35	2	2	4	88	92	15	0.4	5.4
11	C74751	33/M	8200	66	31	3	2	4	82	104	14	0.4	6.8
12	C77242	34/M	4800	56	30	4	2	4	104	118	33	0.8	4.0
13	C77701	28/M	5000	61	34	5	2	4	103	120	14	0.4	7.9
14	C78713	53/F	8000	50	34	6	2	4	70	88	19	0.5	4.0
15	C79434	29/F	6600	38	33	4	2	4	99	111	15	0.4	3.7
16	C78007	38/M	10000	42	28	6	2	4	95	107	17	0.5	6.6
17	C80489	40/M	7800	41	35	24	4	8	99	106	17	0.5	6.2
18	C80919	29/F	7000	72	23	05	2	6	83	106	14	0.4	2.9
19	C81353	60/M	6200	60	34	06	2	8	102	123	18	0.5	6.5
20	C81148	36/M	7200	50	44	06	2	6	96	109	26	0.6	3.1
21	C82243	22/M	5000	34	56	03	4	8	76	102	14	0.4	4.3
22	C81712	60/M	5600	65	30	05	8	18	87	100	14	0.4	4.7
23	C82707	42/M	8200	54	42	04	2	10	86	98	16	0,5	5.3
24	C82990	27/M	7300	64	31	5	2	4	98	109	24	0.7	5.5
25	C84030	38/M	7700	52	26	2	2	4	91	102	22	0.6	5.0
26	C80753	48/M	7100	56	39	5	2	10	94	106	20	0.6	3.8
27	C85766	32/M	7800	55	40	5	2	4	94	108	17	0.5	4.8
28	C85523	25/M	7000	55	40	5	6	12	82	101	15	0.4	5.0
29	C87868	42/F	7700	60	35	5	8	16	96	103	16	0.5	3.5
30	C86025	40/M	8600	38	45	7	2	4	99	108	21	0.7	5.0
31	C88682	45/M	5600	48	32	4	2	4	109	112	15	0.5	5.2
32	C89476	33/F	7600	58	32	3	2	4	82	103	19	0.5	3.8
33	C95276	37/F	8600	74	23	3	4	8	87	98	14	0.4	3.8
34	3888	52/F	8700	53	32	3	5	10	74	90	21	0.8	5.2
35	4965	32/M	7300	66	30	4	12	24	82	102	15	0.6	3.4
36	5055	48/M	6800	66	30	4	10	20	116	123	15	0.5	4.5
37	4061	32/F	10800	76	20	4	8	16	103	124	25	0.5	6.9
38	4099	35/F	10900	52	28	2	6	12	92	109	15	0.3	3.0
39	4156	55/F	8800	67	30	3	8	18	96	102	18	0.6	3.0
40	5115	27/M	6400	44	23	4	4	12	80	93	16	0.4	4.0

CHOLESTEROL PROFILE OF THE OPD AND IPD PATIENTS (BEFORE AND AFTER TREATMENT)

S.NO	OP/IP NO	Age/sex	T.CHOLESTEROL mg/dl		HDL mg/dl		LDL mg/dl		VLDL mg/dl		TGL mg/dl	
			BT	AT	BT	AT	BT	AT	BT	AT	BT	AT
1.	C69642	24/F	151	151	37	30	93	89	21	20	103	106
2.	C71045	31/M	168	112	38	29	109	102	21	21	104	96
3.	C73030	43/M	216	207	59	38	121	108	41	39	129	120
4.	C73050	43/M	232	202	60	35	116	106	36	23	78	75
5.	C72496	32/F	178	111	35	22	105	62	38	30	159	151
6.	C74152	52/M	169	117	36	26	93	44	40	25	120	114
7.	C73299	32/M	180	132	35	29	120	50	25	21	124	106
8.	C73332	42/M	182	156	37	32	118	90	27	19	136	98
9.	C75054	50/F	168	140	43	29	79	62	42	40	134	120
10.	C76543	24/M	191	135	35	30	124	90	12	11	62	57
11.	C74751	33/M	210	186	38	35	115	110	27	23	139	125
12.	C77242	34/M	185	169	32	32	124	106	19	24	98	72
13.	C77701	28/M	186	137	38	30	101	92	42	25	139	127
14.	C78713	53/F	223	219	42	40	122	115	29	23	149	136
15.	C79434	29/F	160	106	35	30	113	63	12	11	61	57
16.	C78007	38/M	180	178	30	36	120	112	30	28	154	143
17.	C80489	40/M	213	198	47	42	112	102	44	28	150	142
18.	C80919	29/F	190	147	36	30	117	108	41	27	106	98
19.	C81353	60/M	209	192	40	42	125	120	39	34	118	112
20.	C81148	36/M	195	190	38	35	109	102	42	40	123	119
21.	C82243	22/M	186	149	36	34	130	119	28	25	129	127
22.	C81712	60/M	150	138	30	30	102	99	34	29	123	107
23.	C82707	42/M	143	136	26	35	94	86	26	25	132	227
24.	C82990	27/M	195	178	38	38	116	108	34	29	134	125
25.	C84030	38/M	95	92	19	30	126	40	16	20	83	81
26.	C80753	48/M	176	168	30	40	100	79	41	32	106	93
27.	C85766	32/M	225	192	45	45	121	119	37	22	158	112
28.	C85523	25/M	176	143	29	31	100	86	42	35	112	108
29.	C87868	42/F	180	154	54	35	110	80	30	28	122	110
30.	C86025	40/M	194	189	29	36	104	86	39	25	149	79
31.	C88682	45/M	144	140	30	28	101	98	34	31	150	115
32.	C89476	33/F	150	148	32	28	90	87	24	23	122	120
33.	C95276	37/F	176	158	35	36	116	96	36	31	164	156
34.	3888	52/F	145	140	36	32	77	75	32	30	161	158
35.	4965	32/M	134	128	21	30	115	90	27	26	137	122
36.	5055	48/M	160	157	24	34	60	61	13	12	67	61
37.	4061	32/F	159	154	32	39	117	98	12	15	62	59
38.	4099	35/F	211	200	40	38	106	104	21	20	107	102
39.	4156	55/F	153	152	32	35	100	96	13	18	66	64
40.	5115	27/M	131	128	30	25	70	64	27	24	139	130

BT – BEFORE TREATMENT AT – AFTER TREATMENT

URINE CULTURE AND SENSITIVITY				
S.NO	**OP/IP NO**	**Age/sex**	**BEFORE TREATMENT**	**AFTER TREATMENT**
1.	C69642	24/F	No growth in culture	No growth in culture
2.	C71045	31/M	No grown in culture	No grown in culture
3.	C73030	43/M	No grown in culture	No grown in culture
4.	C73050	43/M	E-coli Grown in culture	E-coli Grown in culture
5.	C72496	32/F	No growth in culture	No growth in culture
6.	C74152	52/M	E-coli Grown in culture	Proteus species grown in culture
7.	C73299	32/M	No growth in culture	No growth in culture
8.	C73332	42/M	E-coli Grown in culture	**No grown in culture**
9.	C75054	50/F	E-coli Grown in culture	**No grown in culture**
10.	C76543	24/M	No growth in culture	No growth in culture
11.	C74751	33/M	No growth in culture	No growth in culture
12.	C77242	34/M	No growth in culture	No growth in culture
13.	C77701	28/M	Pseudomonas isolated in culture	Pseudomonas isolated in culture
14.	C78713	53/F	Bacterial aerobic culture	**No growth in culture**
15.	C79434	29/F	No growth in culture	No growth in culture
16.	C78007	38/M	No growth in culture	No growth in culture
17.	C80489	40/M	No growth in culture	No growth in culture
18.	C80919	29/F	No growth in culture	No growth in culture
19.	C81353	60/M	No growth in culture	No growth in culture
20.	C81148	36/M	No growth in culture	No growth in culture
21.	C82243	22/M	No growth in culture	No growth in culture
22.	C81712	60/M	No growth in culture	No growth in culture
23.	C82707	42/M	No growth in culture	No growth in culture
24.	C82990	27/M	No growth in culture	No growth in culture
25.	C84030	38/M	No growth in culture	No growth in culture
26.	C80753	48/M	No growth in culture	No growth in culture
27.	C85766	32/M	No growth in culture	No growth in culture
28.	C85523	25/M	No growth in culture	No growth in culture
29.	C87868	42/F	No growth in culture	No growth in culture
30.	C86025	40/M	No growth in culture	No growth in culture
31.	C88682	45/M	Enterococcus faecalis isolated in culture	Enterococcus faecalis isolated in culture
32.	C89476	33/F	E-coli Grown in culture	**No growth in culture**
33.	C95276	37/F	E-coli Grown in culture	**No Growth in culture**
34.	3888	52/F	E-coli Grown in culture	E-coli Grown in culture
35.	4965	32/M	E-coli Grown in culture	E-coli Grown in culture
36.	5055	48/M	No growth in culture	No growth in culture
37.	4061	32/F	No organism grown	No organism grown
38.	4099	35/F	No growth in culture	No growth in culture
39.	4156	55/F	E-coli Grown in culture	Klebsiella grown in culture
40.	5115	27/M	No growth in culture	No growth in culture

LAB INVESTIGATIONS BEFORE TREATMENT													
S.no	OP/IP	Age/ Sex	T.Billirubin mg/dl	D.Billirubin mg/dl	ID.billirubin mg/dl	SGOT U/l	SGPT U/l	SAP U/l	T.Protein Gm/dl	Albumin gm/dl	Globulin gm/dl	Calcium mg/dl	Phosphorous mg/dl
1	C69642	24/F	0.5	0.3	0.2	20	22	131	6.8	5.4	1.4	11.8	3.3
2	C71045	31/M	0.9	0.5	0.4	24	25	115	7.2	5.3	1.9	13.3	2.9
3	C73030	43/M	0.5	0.3	0.2	18	26	127	7.9	5.8	2.1	12.4	5.1
4	C73050	43/M	0.5	0.3	0.2	22	21	230	7.4	5.4	2	10.6	5.9
5	C72496	32/F	0.4	0.2	0.2	19	21	121	7.6	5.5	2.1	11.6	4.2
6	C74152	52/M	0.7	0.4	0.3	17	16	185	6.9	5.4	1.5	11.5	3.6
7	C73299	32/M	0.5	0.3	0.2	25	27	170	6.9	5.4	1.5	11.9	2.6
8	C73332	42/M	0.5	0.3	0.2	21	28	200	6.7	4.8	1.9	11.7	2.8
9	C75054	50/F	0.7	0.4	0.3	20	22	240	7.7	5.1	2.6	12	3
10	C76543	24/M	0.6	0.2	0.4	17	19	202	7.5	4.4	3.1	13.4	3
11	C74751	33/M	0.6	0.3	0.3	22	25	146	7.9	5	2.9	11	3.9
12	C77242	34/M	0.6	0.3	0.3	20	22	128	7	5.1	1.9	12.1	2.3
13	C77701	28/M	0.7	0.3	0.4	23	25	218	7	4.9	2.1	9.6	2.5
14	C78713	53/F	0.6	0.2	0.4	20	21	129	7.6	4.1	3.5	9.1	3.2
15	C79434	29/F	0.6	0.4	0.2	13	15	138	6.5	4.3	2.2	10	2.3
16	C78007	38/M	0.6	0.2	0.4	18	20	269	6.9	4.3	2.6	11	3.7
17	C80489	40/M	0.6	0.2	0.4	20	24	221	7.5	5.1	2.4	11.2	3.3
18	C80919	29/F	0.6	0.2	0.4	24	26	185	6.2	4	2.2	10.2	3.9
19	C81353	60/M	0.8	0.3	0.5	23	28	166	7.1	4.5	2.6	11.2	4.4
20	C81148	36/M	1.2	0.6	0.6	22	21	129	6.8	4.5	2.3	10.8	4

S.no	OP/IP	Age/ Sex	T.Billirubin mg/dl	D.Billirubin mg/dl	ID.billirubin mg/dl	SGOT U/l	SGPT U/l	SAP U/l	T.Protein Gm/dl	Albumin gm/dl	Globulin gm/dl	Calcium mg/dl	Phosphorous mg/dl
21	C82243	22/M	0.6	0.2	0.4	27	23	157	6.9	4.6	2.2	10.9	3.1
22	C81712	60/M	0.5	0.2	0.3	16	18	184	7.1	5	2.1	10.3	2.8
23	C82707	42/M	0.5	0.2	0.3	24	22	260	7	4	3	9.8	3.1
24	C82990	27/M	0.9	0.4	0.5	15	16	186	7.1	4.5	2.6	10.1	3
25	C84030	38/M	0.6	0.2	0.4	21	22	238	7.7	4.8	2.9	10.6	3.1
26	C80753	48/M	0.5	0.2	0.3	16	18	170	7.6	5.6	2	9	3.8
27	C85766	32/M	1	0.6	0.4	25	24	165	7.9	4.7	3.2	11.3	3.6
28	C85523	25/M	0.4	0.2	0.2	24	25	200	7	5	2	10.4	3.6
29	C87868	42/F	0.4	0.2	0.2	24	26	180	7	4	3	10	2.9
30	C86025	40/M	0.5	0.2	0.3	25	23	220	7.1	3	4.1	10.7	2.8
31	C88682	45/M	0.6	0.2	0.4	17	19	149	6.2	4	2.2	10.4	3
32	C89476	33/F	0.5	0.2	0.3	14	15	187	6.9	3.4	3.5	11.4	2.8
33	C95276	37/F	0.6	0.2	0.4	15	17	220	5	3	2	10.8	3
34	3888	52/F	0.4	0.2	0.2	24	21	256	7	4.3	2.7	12.8	2.8
35	4965	32/M	0.8	0.3	0.5	18	20	145	7.8	4.6	3.2	10.4	3
36	5055	48/M	0.4	0.2	0.2	21	22	170	6.6	4.5	2.1	10.6	3.1
37	4061	32/F	1	0.6	0.4	12	10	136	6.6	4.5	2.1	10	3
38	4099	35/F	0.5	0.2	0.3	20	22	159	6.4	4.2	2.2	10.5	3
39	4156	55/F	0.6	0.2	0.4	21	23	168	6.8	4.8	2	12.4	2.8
40	5115	27/M	1	0.5	0.5	20	21	166	6.5	4	2.5	10.9	2.9

LAB INVESTIGATIONS AFTER TREATMENT													
S.no	OP/IP	Age/Sex	T.Billirubin mg/dl	D.Billirubin mg/dl	ID.billirubin mg/dl	SGOT U/l	SGPT U/l	SAP U/l	T.Protein gm/dl	Albumin gm/dl	Globulin gm/dl	Calcium mg/dl	Phosphorous mg/dl
1	C69642	24/F	0.6	0.2	0.4	18	19	190	7	5.2	1.8	11	3
2	C71045	31/M	0.7	0.2	0.5	21	26	216	6	4	2	11.3	2.7
3	C73030	43/M	1.1	0.5	0.6	19	28	119	6.1	3.2	2.9	11	3
4	C73050	43/M	0.4	0.2	0.2	24	24	262	6.8	4	2.8	10.1	4.6
5	C72496	32/F	0.5	0.2	0.3	15	16	121	7	5	2	11	3
6	C74152	52/M	0.4	0.2	0.2	21	28	190	6.9	3.9	3	10	3
7	C73299	32/M	0.3	0.2	0.1	16	17	195	7.4	4.4	3	10.6	2.8
8	C73332	42/M	0.6	0.2	0.4	21	24	211	6.5	4.5	2	11	3.6
9	C75054	50/F	0.5	0.2	0.3	19	20	152	7.7	5.7	2	12.2	3.6
10	C76543	24/M	0.5	0.2	0.3	23	26	236	6.4	3.4	3	12.2	2.8
11	C74751	33/M	0.7	0.3	0.4	16	20	186	7.5	5.5	2	10.5	3
12	C77242	34/M	0.5	0.2	0.3	22	27	196	7.5	5.5	2	9.3	2
13	C77701	28/M	0.6	0.2	0.4	24	23	213	6.7	4.2	2.5	11	3.2
14	C78713	53/F	0.5	0.2	0.3	12	14	147	6.6	4.1	2.5	10.1	3
15	C79434	29/F	0.4	0.2	0.2	11	12	145	6	3.6	2.4	9.8	2
16	C78007	38/M	1.1	0.5	0.6	19	25	176	6.6	3.7	2.9	10.8	3.1
17	C80489	40/M	0.4	0.2	0.2	11	23	177	6	3.6	2.4	9.9	2.7
18	C80919	29/F	0.5	0.2	0.3	16	26	186	6.6	4.1	2.4	10.1	3.5
19	C81353	60/M	0.5	0.2	0.3	16	17	183	5.6	3.1	2.5	10.8	3.5
20	C81148	36/M	1	0.5	0.5	18	22	220	6.2	4.2	2	9.2	3.8

S.no	OP/IP	Age/Sex	T.Billirubin mg/dl	D.Billirubin mg/dl	ID.billirubin mg/dl	SGOT U/l	SGPT U/l	SAP U/l	T.Protein gm/dl	Albumin gm/dl	Globulin gm/dl	Calcium mg/dl	Phosphorous mg/dl
21	C82243	22/M	0.7	0.3	0.4	23	26	176	6	4	2	9.8	2.8
22	C81712	60/M	0.7	0.3	0.4	19	20	156	6.4	4.4	2	9.6	2.6
23	C82707	42/M	0.6	0.2	0.4	23	25	170	6.9	4.3	2.6	10.1	3
24	C82990	27/M	1	0.7	0.3	22	27	166	6.5	4.4	2.1	10.8	3
25	C84030	38/M	0.5	0.2	0.3	22	24	198	6.2	4	2.2	10.8	2.9
26	C80753	48/M	0.5	0.2	0.3	24	26	179	6.5	4.5	2	11	3.2
27	C85766	32/M	1	0.5	0.5	23	26	168	5.9	2.6	3.3	10.2	2.9
28	C85523	25/M	0.9	0.3	0.6	16	18	144	5	3	2	10	2.9
29	C87868	42/F	0.5	0.2	0.3	19	21	149	6.6	3.6	3	10.4	2.9
30	C86025	40/M	0.8	0.3	0.5	24	26	242	6.6	4.4	2.2	11.1	3.3
31	C88682	45/M	0.6	0.2	0.4	16	14	135	6	4	2	9.6	2.3
32	C89476	33/F	0.4	0.2	0.2	13	15	164	6.2	4.2	2	10.2	2.5
33	C95276	37/F	0.6	0.2	0.4	13	14	135	5.9	4.5	1.4	9.7	2.9
34	3888	52/F	0.4	0.2	0.2	23	21	220	6.4	4.4	4	10.2	2.4
35	4965	32/M	0.8	0.3	0.5	18	20	145	7.8	4.6	3.2	10	3
36	5055	48/M	1.2	0.6	0.6	16	17	161	7	5	2	10.4	2.9
37	4061	32/F	3.6	1.2	2.4	12	14	150	6.5	4.1	2.4	10.2	2.9
38	4099	35/F	0.5	0.2	0.3	18	16	154	6.2	4.2	2	9.6	2.8
39	4156	55/F	0.5	0.2	0.3	11	12	140	6	3	2	10.8	3.3
40	5115	27/M	0.8	0.4	0.4	18	17	160	6.4	4	2.4	9.8	2.5